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CLINICAL CARDIOLOGY



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CLINICAL CARDIOLOGY

BY

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TO
MY PARENTS
IN TRIBUTE TO THEIR CONSTANT SELF-
SACRIFICE, THIS BOOK
IS AFFECTIONATELY DEDICATED

44191

PREFACE

IN the many recent books on cardiac disease, dealing chiefly with polygraphy, electrocardiography, and orthodiascopy, there has been much confusion regarding the proportionate value of graphic methods, with the result that undue emphasis has been placed upon purely instrumental and technical considerations. The author has, therefore, included in this book the graphic as well as the usual bedside methods, while writing from the clinician's, rather than from the cardiologist's standpoint, and he believes that this work will therefore supply a comprehensive, practical reference book for both practitioner and student.

The opening chapters are devoted to a description of instrumental and graphic methods in the study and examination of normal and abnormal rhythms and of normal and abnormal silhouettes. From a study of these chapters, the physician is enabled to discern the relation and application of instrumental methods to clinical cardiology and bedside examination. Without minimizing their importance, I have emphasized the fact that instrumental methods are not infrequently subsidiary. Indeed, as will be shown, graphic devices may sometimes be dispensed with if their fundamental significance is comprehended.

Subsequent chapters are devoted to the purely clinical side of cardiology. Careful consideration is given to the important subjects of the pathology, etiology, diagnosis, prognosis, and therapy of endocarditis, myocarditis, and cardiosclerosis. Questions occurring in everyday practice with reference to diet, exercise, and general management of heart disease are fully dealt with. There are special chapters on precordial pains, blood pressure, and the heart in pneumonia.

I desire to express my sincere thanks to Dr. Alfred E. Cohn of the Rockefeller Institute for valuable aid and suggestions, especially on the chapters dealing with the arrhythmias; to my publishers, The Macmillan Company, for courteous coöperation; and to my wife, without whose aid and stimulus this book could not have been written.

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CLINICAL CARDIOLOGY

CHAPTER I

THE HEART

Development of the Heart. — In very early fetal life the heart is represented by a simple tube, the **cardiac tube**. It consists of two distinct strata: an inner, thinner layer, derived from the hypoblast, which later becomes the endocardium; and an outer, thicker layer, derived from the visceral mesoblast, which finally develops into the musculature. Later, the cardiac tube elongates and becomes bent upon itself so as to form an S-shaped loop, with an anterior right, and a posterior left angle. Slight constrictions soon appear, which serve to divide the loop into four parts (Fig. 1): (1) the sinus venosus (sinus reuniens of His), (2) the common auricle, (3) the common ventricle, (4) the aortic bulb. The **sinus venosus** is at first situated transversely behind the common auricle and connects with the latter by a median aperture; it afterwards becomes oblique and divides into two projections or horns: the right forms part of the right auricle, the line of union being marked in the adult heart by a vertical crest, the crista terminalis of His; the left horn persists as the coronary sinus. The **common auricle** (*C.A.*) becomes partitioned off into right and left auricles by the gradual formation of a septal wall, the septum superius; the foramen ovale results from a perforation of this wall. Another septal structure, the septum inferius, similarly forms, and separates the **common ventricle** (*C.V.*) into right and left chambers. For some time, however, it does not quite reach the auricular canal, thus leaving a foramen between the auricles and ventricles, the common auriculo-ventricular orifice. The formation of a septum is also responsible for the division of the **aortic bulb** (*A.B.*) into the two great vessels, — the aorta and the pulmonary artery.

Position of the Fetal Heart. — In early fetal life, the heart lies immediately under the head and is of relatively large size. Later, it becomes a thoracic organ, lying at first vertically, then gradually assuming

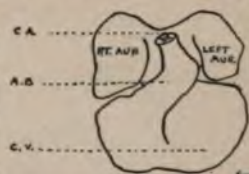


FIG. 1. — Heart of the human embryo 5 mm. long. Front view. $\times 30$. (Modified from His.)

a more oblique position. The auricular portion with its intercommunication (the foramen ovale) is at first larger than the ventricle. By means of the ductus arteriosus (ductus Botalli), the blood from the right ventricle and pulmonary artery passes mainly to the aorta instead of to the lungs. To carry on this circulation, the wall of the right ventricle is correspondingly muscular and as thick as that of the left. Toward the end of fetal life, the left ventricle becomes thicker and heavier than the right.

Weight of the Adult Heart and Position of the Valves. — The average normal adult heart weighs, in the male, from 280 to 360 grams ($9\frac{1}{2}$ to 12 oz.), in the female, from 240 to 330 grams (8 to 11 oz.); its

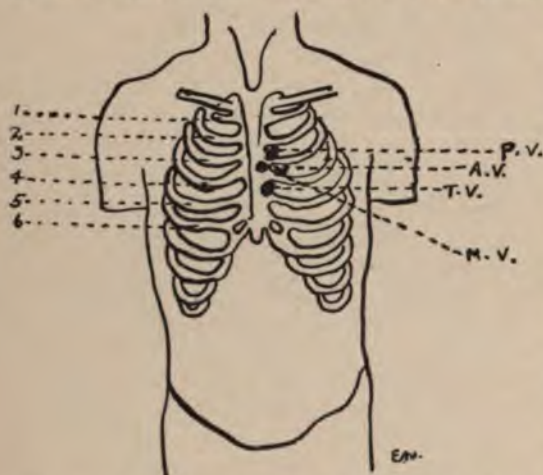


FIG. 2. — Diagrammatic position of the cardiac valves.

P.V = pulmonary valves; T.V = tricuspid valves;
A.V = aortic valves; M.V = mitral valves.

proportion to body weight ranges approximately from 1:160 to 1:170. Though hearts vary considerably in size within normal limits, the average length of the adult heart on its longest axis is from twelve to fifteen centimeters, its greatest breadth, nine to eleven centimeters. The adult heart lies behind the lower two thirds of the sternum. The methods for determining the position of the valves with the heart *in situ* by frozen sections, and also by coating the valvular surface with lead and subsequently taking radiographs, have not given uniform results. According to Piersol, the **aortic valves** lie behind the left half of the sternum, a little below and to the right of the pulmonary valves; the **latter** are situated behind the sternal end of the third left costal cartilage. The **tricuspids** are situated behind the midsternum, opposite the fourth interspace and the fifth chondro-sternal articulation; the **mitral valves** are opposite the sternal end of the third left interspace (Fig. 2).

Anatomy and Physiology of the Heart. — The heart consists of three layers: the endocardium, pericardium, and myocardium. The **endo-cardium** is a connective tissue membrane containing smooth muscle and numerous elastic fibers. Its free endocardial surface consists of a single layer of irregularly shaped polygonal endothelial cells. The **epi-cardium** is a connective tissue structure containing fat cells and elastic fibers; its outer surface is covered with a single layer of squamous

epithelium. The **muscle fibers** of the heart although transversely striated, must be regarded, developmentally and histologically, as modifications of smooth muscle. The undifferentiated protoplasm of the heart muscle fiber — the sarcoplasm — is found chiefly in its axial part. The oval nucleus, which frequently contains oil droplets or pigment granules, is embedded in the sarcoplasm. The muscle cells anastomose by means of short, oblique, or transverse processes into various complexes and layers. In the **auricular musculature** there is a superficial muscular layer which runs transversely and encircles both auricles. Each auricle also possesses a relatively independent system of fibers which runs at right angles to the superficial layers. The **course of the ventricular fibers and layers** is quite complex and as yet a matter of uncertainty. According to the most reliable studies, the fibers on the ventricular surface arise from tendinous rings and membranes at the base of the heart; here they form a vortex, pass into the interior of the left ventricle to the septum, and connect with the papillary muscles; they thus turn on themselves toward the base and form spiral loops, which when contracting, approximate base and apex, and at the same time rotate the apex clockwise from left to right. Mall divides these "superficial fibers" into two groups: the **Superficial Bulbo-spiral** and **Superficial Sino-spiral**. The former belong chiefly to the left ventricle. They arise from the conus to the left of the aorta and left ostium venosum, proceed spirally, penetrate to the interior of the left ventricle and end in the septum and posterior aspect of the ventricle; at that point they connect with the posterior papillary muscles. Some of the deeper fibers of this layer encircle the lower part of the ventricle and pass upward, to end at the base of the heart. The superficial sino-spiral fibers arise mainly from the posterior aspect of the heart in the neighborhood of the right ostium venosum, proceed spirally (though more transversely than the first group) to the apex over the anterior surface of the right ventricle. They penetrate the interior of the left ventricle and terminate on its anterior surface and in the papillary muscles, especially the anterior. Beneath the superficial layers of the bulbo- and sino-spiral systems lie similar **deep layers** which run more transversely or circularly. The **deep bulbo-spiral** layer encircles the left ventricle and ends by way of the septum on the dorsal side of the aorta. Some fibers make a circular loop around the conus at the base of the pulmonary artery. The entire layer makes a strong circular system whose contraction tends to diminish the lumen of the left ventricle. The **deep sino-spiral** layer originates from the posterior aspect of the left ostium venosum, passes transversely to enter the interior of the right ventricle, and then turns upward toward the base. Here some strands pass circularly around the base of the heart and left ostium.

The **arterial supply** of the heart is derived from the right and left coronaries. Though often so regarded, they are not end arteries, for anastomoses have been clearly demonstrated by Spalteholz's method.

Furthermore, blood is sometimes found beyond an old, complete obstruction of the coronary artery. The veins accompany the arteries and empty in the right auricle. The **lymph vessels** are very abundant; they are formed from radicals derived from the lymph spaces in the clefts between the muscle fibers; they accompany the blood vessels in their course and terminate in the thoracic and right lymphatic ducts.

Attributes of the Cardiac Musculature. — Engelmann and Gaskell, by careful experimentation some thirty-five years ago, established certain attributes of the cardiac musculature, namely: **irritability, contractility, rhythmicity, and conductivity**. These have been respectively designated by Engelmann as bathmotropic, inotropic, chronotropic, and dromotropic functions; he has qualified them as positive or negative, depending upon influences which act favorably or unfavorably upon the individual functions. This nomenclature, however, has not found its way in general clinical use. To the four attributes mentioned, Gaskell has added a fifth — **tonicity**. This property is similar to that found in ordinary skeletal musculature, which, in the case of the heart, keeps it in a slight state of contracture even during diastole. Tonicity is a term which is perhaps used too loosely and indiscriminately and is often confused with the contractile power of the heart; it is the attribute about which few clinical or experimental facts are known.

It is necessary to emphasize not alone the differentiation of the various cardiac properties, but the fact that certain parts of the heart are endowed with these properties in varying degrees; for example, the sinus region with rhythmic attributes.

Regarding our present knowledge of these various functions, it may be stated that though much is known about disturbances of rhythm and conductivity, many details of the other properties are still wanting.

Besides the cardiac properties mentioned, another is that each **systole** is of **maximum intensity**. This property — the so-called all- or none-reaction — means that the heart muscle answers any stimulus sufficient to cause response by a **maximal contraction**. The latter is probably due to the intricate intertwined cardiac muscular system already described. Cardiac contractility therefore does not depend upon the strength of the impulse; it varies, however, considerably with the state of irritability, one of the heart muscle functions. Regarding this function, during systole the heart is not irritable, it is **refractory** to all other impulses and therefore cannot be tetanized like skeletal musculature. There is an experimental exception to this law; hearts that have been poisoned by muscarine, alcohol, chloral, etc., may have a shortened refractory phase and hence may be brought into tetanic contraction by proper stimuli.

Nerve Supply. — Situated at the base of the heart are the intercommunicating **superficial** and **deep cardiac plexuses**, from which the extrinsic cardiac nerves are derived. The superficial plexus lies in the

concavity of the aortal arch; the deep, between the trachea and aorta. The latter plexus is composed of nerves derived from the sympathetic cervical ganglia and the cardiac branches of the recurrent laryngeal and vagus. The branches from the right side of the plexus go to form parts of the anterior and posterior coronary plexuses, besides sending a few filaments to the right auricle. The branches from the left side are distributed to the left auricle and compose a large part of the posterior coronary plexus. The superficial cardiac plexus forms the chief part of the anterior coronary plexus.

The posterior and anterior coronary plexuses surround and accompany the branches of the right and left coronary arteries, respectively, and distribute filaments to the ventricular musculature. The coronary plexuses as well as their muscular filaments are richly supplied with ganglia.

Intracardiac ganglia have been found in the auricular wall, at the entrance of the superior and inferior vena cava and at the mouth of the coronary sinus. They have also been found at the level of the auriculo-ventricular junction, especially about the aorta and the pulmonary artery. Scattered ganglia have likewise been observed in the upper part of the ventricles. One observer claims to have discovered ganglion cells along the entire ventricular chamber.

The nerves of the conduction system are described in the next chapter.

The **extrinsic nerves** to the heart are derived from the cardiac plexuses. The heart is often profoundly influenced by impulses which reach it from these nerves. The vagi contain the inhibitory fibers. If, in the experimental animal, the vagi be cut in the neck, the cardiac rate is increased. If the peripheral ends of the cut vagi be stimulated, there is slowing or stoppage of the heart, or a condition in which the auricles beat more rapidly than the ventricles with no rhythmic relationship between the two (Complete Heart Block, Chapter VII). In warm-blooded animals there is not only a diminution of cardiac rate, but also of the strength of auricular and ventricular contraction, until the heart finally stops in diastole. On the other hand, stimulation of the cut end of the sympathetic produces a varying degree of cardiac acceleration. Depending upon the degree of stimulation of vagus and sympathetic, the one or other nerve has a predominating influence upon the cardiac rate. Though antagonistic, this antagonism cannot be measured purely arithmetically; if vagus influence predominates during the course of the experiment, after cessation of stimulation, a typical accelerator influence results. This fact in itself indicates that a certain amount of **tone** is present in both nerves. Nerves of sensation have not been discovered in the heart. Since the lower cervical and upper dorsal nerves, which supply the integument of the neck, chest, and upper extremity, also send filaments to the deep and superficial cardiac plexuses, painful skin areas of varying degrees of intensity and extent are found

as the result of reflex excitation originating in the heart itself from some pathological process or function (Chapter XXI).

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CHAPTER II

THE CONDUCTION SYSTEM

Position and Structure of the Pacemaker. — Thorough experimental electrocardiographic investigations in mammals, and histological and pathological studies in man and animals, have proven that the *primum movens* — the normal impulse center in the human being — exists in a bit of specialized cardiac tissue situated in the sinus region immediately beneath the epicardium and in the groove between the right auricle and superior vena cava. This rhythm center is variously known as the **pacemaker**, the **sino-auricular node (S-A node)**, or the **node of Keith-Flack**. Its shape is irregularly pyriform (Fig. 3) with a larger upper, and a somewhat tapering lower end. The node is surrounded by branches of the vagus and sympathetic; it contains a

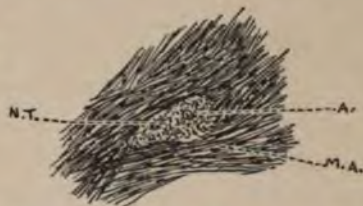


FIG. 3.—Schematic view of sino-auricular node of dog, showing general form of the node and differential structure. A = artery; N.T. = nodal tissue; M.A. = auricular musculature.

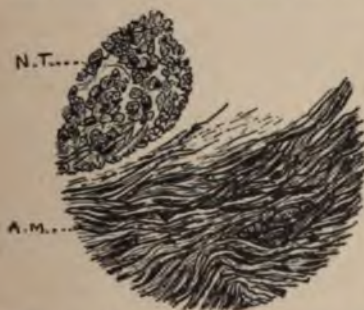


FIG. 4.—Detail of the S-A node of dog (high power), showing the cells. N.T. = nodal tissue; A.M. = auricular musculature.

plexus of moniliform nerve fibrils and a few ganglionic cells; it is therefore, histologically considered, a **neuro-muscular structure**. Its blood supply is derived from a special artery. The structure, arrangement, and composition of the node differ materially from the remainder of the cardiac musculature. The cells are smaller, stain more delicately, and are paler; the cross striations are indistinct or may be absent, the nucleus is pale, and there is a relative richness of perinuclear sarcoplasm. The cells contain more glycogen than those of the non-specialized muscle. They do not follow any orderly layer-like arrangement, but are placed irregularly in a rich stroma of fine connective tissue (Fig. 4). A small specialized muscle band con-

necting the sino-auricular node with the remainder of the conduction system has been described by Thorel, but its presence has not been corroborated by other observers.

Position and Distribution of the Auriculo-ventricular Conduction System. — Similar in structure to the sino-auricular node is the larger

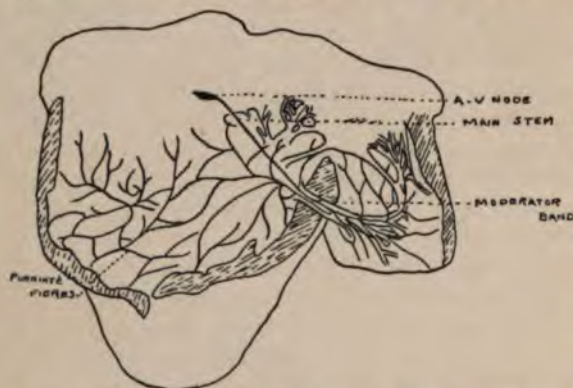


FIG. 5. — Right ventricle of ox heart, showing the auriculo-ventricular node (A-V node) and the main stem. The right branch is seen running along the "Moderator Band." (Modified from Tawara — "Das Reitzleitungssystem des Säugethierherzens.")

mass of specialized tissue, known as the **atrio- or auriculo-ventricular conduction system, or junctional tissue.** For purposes of anatomical identification, its various parts are differently named, but they together form one continuous strand. The **node of Tawara** — the beginning of the junctional tissue — and the **bundle of His** constitute the major

part of the conduction system before its division. They lie immediately beneath the endocardium in the lower part of the right auricle, slightly above the level of the ventricle, and about midway between the opening of the coronary sinus and the fibrous tissue beneath the aortal cusps (the aortal vestibule or "undefended space"). The conduction system is then continued into the **main stem**, which soon makes a hairpin-like division into its two main branches — the **right** (Fig. 5) and **left** (Fig. 6). Both branches course on either side of the interventricular septum in a direction roughly parallel to the axes of the respective ventricular cavities, and are contained in their own sheaths. The right branch is thin and spreads out in a somewhat fan-shaped fashion; the left is more compact, thicker, and club-like. In their

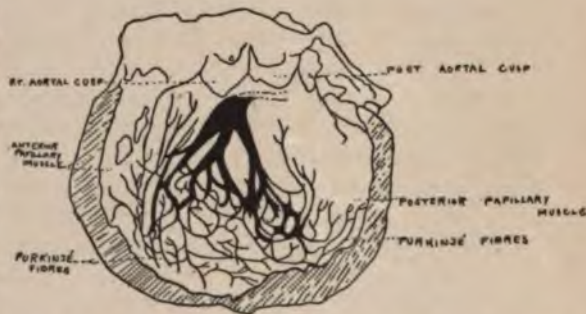


FIG. 6. — Left ventricle of ox heart, showing conduction system. (Modified from Tawara.)

upper portions, both branches are superficial and subendocardial. They split into secondary and minor branches as they spread toward the apex (Figs. 5, 6). In the ox heart, the left branch has three main divisions — to the anterior and posterior papillary muscles, respectively, and to the apex. The right branch has one division that goes to the large papillary muscle and venous base, the other forms the “moderator band” which supplies the septal papillary muscle and the arterial base (conus arteriosus); from both of these, a division to the apex is formed from separate heads. The branches in human and mammalian hearts finally divide into **terminal arborizations** (known as Purkinje fibers in the mammal), which ramify through the papillary muscles and probably throughout the entire ventricular musculature. Some of these terminal arborizations run free across the apical portion of the ventricular cavities; they were formerly regarded as aberrant tendinous strands. They are macroscopically distinguished from the latter by their paler and finer structure and non-glistening appearance. The main stem and branches of the conduction system are sometimes dimly distinguished through the shimmering endocardium as paler, non-glistening structures.

Dissection and Demonstration of the Conduction System. — In order to dissect out the conduction system, the endocardium over the site of the auriculo-ventricular (*A-V*) conduction system is gently teased off with forceps after the heart has been washed in water and hardened in a formaldehyde solution. The conduction system is most readily dissected in the ox or calf heart, less readily in the human. The heart is first incised with a scalpel near the margin of the inter-ventricular septum. The ventricles are then cut parallel to the latter. In this manner they can be turned back, and the septal walls of the auricle and the interventricular septum exposed without injury to the conduction system. In oxen the main branch on the right side exists as a separate strand — the moderator band. After exposure by teasing in the manner described, the structure of the *A-V* conduction system will be found paler, softer, and more delicate than that of the surrounding musculature. Another method of gross demonstration of the conduction system is by **subendocardial injection** of the main branches with a fine hypodermic needle and syringe containing a 50 per cent solution of India ink. If carefully performed, not only the main branches but the subsidiary divisions stand out prominently as darker strands. In this manner, also, corroborative evidence is derived that ventricular contraction begins at the papillary muscles, for the ink may be seen to reach the latter first and then the base and apex of the heart.

The **cellular structure** of the main junctional tissue is similar to that of the *S-A* node. The arborizations consist of larger, paler cells with large nuclei and with protoplasm containing faint striations at the periphery.

Arterial and Nerve Supply of the *A-V* System. — The main arterial supply of the *A-V* system is derived from a special branch of the right

coronary. The junctional tissue is supplied and intertwined with medullated nerve fibrils and ganglia. There are numerous ganglion cells, — mono-, bi-, and multipolar, — whose processes pass to adjacent ganglion cells, to nerve fibers in the bundle, or through the *A-V* system, some of which end in ganglia cells of the bundle or in the muscle plexus. There is an intricate plexus of varicose fibrils around and in close relation to the muscle fibers of the bundle. Thus the *A-V* system, like the sino-auricular node, is a **neuro-muscular spindle**. Though the exact distribution to the nodes of the extrinsic cardiac nerves is not known, it appears probable that, regarding the vagus distribution, the *S-A* node is supplied chiefly by branches of the right, and the *A-V* by those of the left vagus. There appears to be a similar distribution to the *S-A* and *A-V* nodes by the right and left accelerators, respectively.

Course of the Normal Impulse. — From a study of the physiological and anatomical distribution of the specialized tissues, it is evident that the normal impulse arises in the *S-A* node; it spreads thence through the auricle, following, as far as known, no especially differentiated path in the latter. It then reaches the junctional tissue, and by way of the right and left branches and terminal arborizations, it finally spreads to the papillary muscles and throughout the ventricular musculature.

Myogenic or Neurogenic Impulse? — The question whether the original impulse is of myogenic or neurogenic origin has been for years a matter of dispute, and has not yet been decided. The intimate and intricate relationship existing between the muscle and nerve structures in the nodal regions demonstrates how difficult must be the final determination of this question. It is known clinically that the rhythm center is readily influenced by purely neurogenic impulses; these can indeed upset the normal cardiac control and give rise to abnormal rhythms (Chapter VIII). How far such clinical observations can be applied to the question of the normal control of the rhythm center it is impossible to state. All in all, it seems probable that the sino-auricular node is activated by both neurogenic and myogenic influences, though we have at present no means of discovering under what circumstances either becomes the controlling factor.

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CHAPTER III

POLYGRAPHIC TRACINGS

Mackenzie Ink Polygraph. — For purposes of exact study of arterial and venous pulsations, mechanical methods, instruments, and graphic representation (**sphygmograms**) are necessary.

Instruments that simultaneously transcribe arterial and jugular pulsations are called **polygraphs**. For clinical use I have found the Mackenzie Ink Polygraph perfectly satisfactory. Its compact size, the comparative simplicity and ease of operation, the fact that if necessary very long records can be taken, and the use of ink and a paper roll instead of smoked paper, make it suitable for clinical work.

The Mackenzie polygraph (Fig. 7, Plate I) consists essentially of a clockwork (*Cl*) incased in a metal container. The clock is fitted with a time-marker (*Ti*) which ticks and marks fifth seconds by means of a small pen. The speed of the time-marker may be regulated by a small screw (*S*). There are two separate keys (*S.B* 1, *S.B* 2) for winding the driving gear and the time-marker. There is also a small lever (*Le*) which starts and stops the driving mechanism. Attached to one side of the case is a slot (*Sl*) into which is fitted a support for a paper roll (*Pr*). On the opposite side is a smaller slot, which acts as a rest for a long, narrow arm (*Ar*) for the support of two transmitting tambours (*T.T*); the latter are so arranged that they can move in any direction. To the tambours are attached long writing pens (*P*); the pressure of the points upon the paper may be regulated by manipulating the tambours. Each tambour is also separately connected with the receiving apparatus applied to the venous and arterial pulse by means of elastic tubing (*E.T*). The receiver for the venous pulsations is a small circular metal cup (*Cu*), which may also be used for registering cardiac pulsations by placing it over the apex. For transmitting radial pulsations, a perforated leather strap (*L.S*) is buckled about the wrist and so adjusted that the button (*Bu*) or pelotte rests upon the most prominently pulsating part of the radial; the pressure of the pelotte upon the latter is regulated by a small flat spring (*Sp*). There is a broad wrist tambour (*W.T*) which rests upon the button and transmits radial pulsations to the writing pens through the transmitting tambour.

PLATE I



FIG. 7. — Mackenzie Ink Polygraph.

- Cl* = Clockwork;
- Ti* = Time-marker;
- S* = Screw which regulates speed of time-marker;
- S.B. 1, 2* = Keys for winding the driving gear and time-marker;
- Le* = Lever for starting and stopping the driving mechanism;
- Sl* = Slot for support of the paper roll (*P.R.*);
- Ar* = Arm for the support of the two transmitting tambours (*T.T.*);
- P* = Writing pens;
- E.T* = Elastic tubing connecting the transmitting tambours to the receiving apparatus;
- Cu* = Cup for receiving venous or cardiac pulsations;
- L.S* = Leather strap for buckling over the radial artery;
- Bu* = Button for receiving the radial pulsations;
- Sp* = Screw for regulating the pressure of the button upon the radial;
- W.T* = Wrist tambour.

Method of Use of the Mackenzie Polygraph.—The polygraph is used as follows: the driving mechanism and time-marker are wound up, the paper roll set in place, the pens thoroughly inked and lightly adjusted upon the paper. After palpating the radial, its most pulsatile point is marked by an ink spot, or preferably by two rectangular lines (Γ), one along the radial, the other across the wrist; these serve as guides for the proper position of the pelotte and wrist strap. The best position of the wrist is with the hand in moderate extension or hyperextension, because this tends to make the radial artery more superficial. This position can be conveniently maintained by firmly pressing the extended hand against the physician's thigh. The upper strap of the wrist attachment is put on loosely so as not to obliterate the artery; the lower is buckled on firmly. The spring regulating the pressure of the pelotte is then pressed down sufficiently to make the latter bob vigorously with the radial pulsations. The wrist tambour is slipped in position with its screw support loose, so that the metal tip on the under surface of the tambour rests full upon the bobbing button; it is then screwed and held in this position. Thus through the receiving and transmitting air system of tambours and their connecting elastic tubing, the radial pulsations are transmitted to the writing pens.

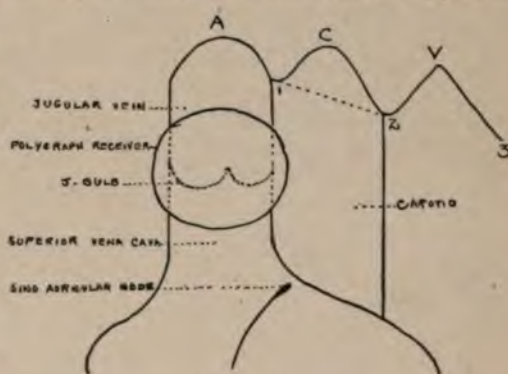


FIG. 8.—Schematic view of the a-c-v waves and of the jugular bulb.

To transmit and transcribe venous pulsations the metal cup is placed over the jugular bulb (Fig. 8), preferably on the right side, because the vein is usually more prominent on that side. The neck of the cup is grasped between the fore and middle fingers, the rim by the thumb, and the cup slid along the outer border of the sterno-mastoid muscle until it touches the clavicle. It thus rests over the triangular area formed by the jugular vein (with its bulb), the inner end of the clavicle, and the sterno-mastoid muscle. The patient is made to lie as flat as possible; he should breathe quietly, for stertorous breathing interferes with proper registration. Rigidity of the neck muscles also mars pulsations. Superabundance of fat and respiratory dyspnoea are other factors which may interfere with or vitiate accurate registration.

After the wrist tambour and venous cup have been satisfactorily adjusted, the pens are separately slid across the paper, so as to establish coincident ordinates for measurement of the curves. These lines need

not necessarily be continuous, for simultaneous venous and arterial pulsations may be measured off and standardized by means of calipers. The driving mechanism is now released and arterial and venous pulsations simultaneously registered.

Correlation between Experimental Auricular Pressure Curves and the Human Jugular Pulse. — Early experimenters gained information regarding auricular pressure curves by inserting sounds directly into the auricles of dogs and horses; they found that auricular contractions were accompanied by increased auricular pressure, that is, by positive pressure waves. While there is a general correspondence between such pressure curves and human jugular tracings obtained by the polygraph, it must be remembered that the latter primarily depend upon difference in volume created in the confined air space of the cup resting over the jugular bulb. These **volumetric differences** are transmitted to the tambour, and the pen is then correspondingly deflected. Hence differences in auricular pressure are not necessarily transmitted and transcribed as volumetric waves; and as a corollary, one can rarely predicate and estimate auricular pressure by the excursion and direction of the "venous" polygraphic waves.

The Waves of the Normal Jugular Pulse, — the Normal Phlebogram. — Frequent observations have shown that, corresponding to the

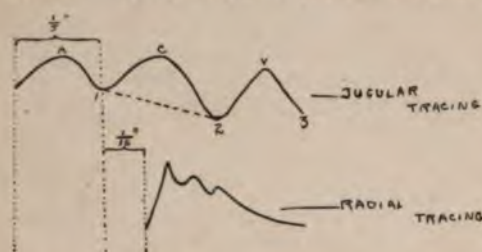


FIG. 9. — Diagrammatic representation of the normal polygraphic curve.

a = auricular wave;
c = carotid wave;
v = ventricular filling wave. (See text.)

first auricular pressure curve coincident with auricular systole, there exists normally a venous pulse best seen in the venous trunks at the root of the neck. In man, for each radial beat, there are in the normal jugular tracing **three waves or elevations**; each elevation is accompanied by a corresponding **depression** (Fig. 9). We shall follow the simple nomenclature usually adopted in the litera-

ture and call the elevations the *a-c-v* waves. The *a* refers to the **auricular**, *c* to the **carotid**, and *v* to the **ventricular filling** wave. The **rise** (Figs. 8-9) and **fall** (Figs. 8-9, 1) of the first wave *a* are caused by the reflux wave produced in the veins of the neck by auricular systole. In rhythmically beating hearts, the *a* wave comes before the advent of ventricular systole (*c* wave). The absence of the *a* elevation in those types of arrhythmia in which experimental and electrocardiographic evidence shows absence of normal rhythmic auricular contractions (Auricular Fibrillation, Chapter VII), and its abnormal position in arrhythmias affecting relationship between auricular and ventricular contractions (Heart Block, Chapter VII), are con-

firmatory evidence that the *a* wave is in the main, if not entirely, due to auricular systole. If the jugular tracings were quite similar to the experimental auricular pressure curves, following auricular systole (*a* wave) there should be a continuous fall (Fig. 9, 1-2) and two elevations (*a-v*) and depressions (2-3) in the venous tracing. The post-auricular fall is interrupted by the advent of an elevation, the *c* wave. For the present disregarding the latter, the chief cause of the postauricular fall of pressure (Figs. 8-9, 1-2) is undoubtedly auricular relaxation following systole; ventricular systole acts as a contributory cause in increasing this relaxation, mainly by dragging down the interventricular septum, and to a lesser degree, by producing diminished intrathoracic pressure. The physiological limit of the duration of the *a* wave is one fifth of a second.

The cause of the **carotid wave** is still a matter of dispute. Its occasional appearance one twentieth of a second before the onset of carotid pulsation, and its presence after experimental ligation have been offered as evidence that the *c* wave is not due to carotid pulsation. However, those who have worked with the cup receiver over the jugular bulb will have observed how the venous tracing is often vitiated by placing the receiver too close to the carotid, in consequence of which the *c* wave will often obtrude itself upon the venous tracing. Although there exists some dispute as to its cause, the practical importance of the incidence of the *c* wave in the study of the human phlebogram rests upon the fact that its foot point is coincident with the onset of carotid pulsation. The **determination of the foot point**, therefore, becomes an important landmark in the study of the polygram. Since the pulse wave reaches the wrist approximately one tenth of a second after its arrival at the carotid artery, and the onset

of the individual radial beats is readily discernible in the arteriogram, the foot point of the *c* wave may be determined and distinguished from the *a* and *v* waves by measuring with calipers from coincident ordinates in the radial and venous tracings. These ordinates are derived by stopping the driving mechanism for a moment and sliding both pens across the paper (Fig. 10); when measuring in the direction of the physiological progression of the waves, one tenth of a second (the difference in time between carotid and radial pulsations) is added; in this manner the foot point of the *c* wave is derived. With the latter determined, it becomes a simple matter in good curves to determine the

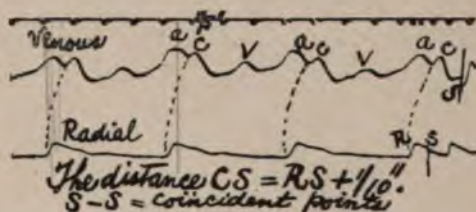


FIG. 10. — Normal venous and radial tracing showing rhythmical *a-c-v* waves in the jugular tracing, and regular radial beats. The points *S-S* are coincident ordinates derived by sliding the pens when the driving mechanism is at rest. The method of derivation of the foot point of the *c* wave is also shown.

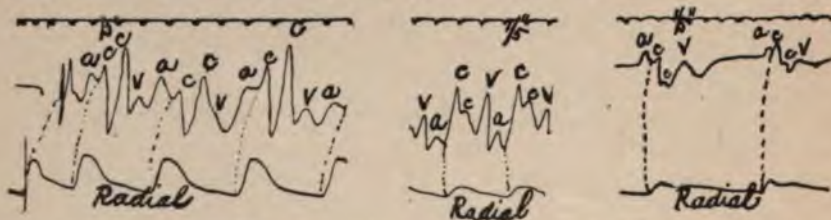
a and *v* waves when the pulse beats rhythmically: the precarotid is the *a*, and the postcarotid, the *v* wave.

The **third positive** (Fig. 9, *v*), the **ventricular wave** which follows the second depression, is due to ventricular systole; it results almost entirely from reflux of the stored auricular blood which is forced into the venous system with ventricular systole while the auriculo-ventricular valves are still closed. Another factor in its production is the sudden release of the base of the ventricle at the commencement of ventricular diastole. The slightly variable beginning of the *v* wave depends upon the varying amount of blood stored up during auricular diastole; its termination is coincident with the opening of the tricuspid valves.

The **third fall or depression** (Fig. 9, 3) is caused by the rapid drop of pressure in the auricle and in the venous trunks at the root of the neck, which follows the beginning of ventricular diastole and the opening of the auriculo-ventricular valves.

Translating the knowledge gained from the graphic orderly sequence of auricular and carotid pulsations (the *a* and *c* waves) into terms of auricular and ventricular systoles, we have the means of studying rhythmical sequential cardiac cycles—the normal pulse—as well as the numerous disturbances of rhythm affecting auricle and ventricle.

Variations in the Normal Phlebogram.—There are certain variations in the normal phlebogram which require consideration. The *a*



FIGS. 11, 12, 13. — These figures show a split *c(c-c)* wave in the venous tracing.

wave is occasionally bifurcated at its apex, *i.e.* at the height of auricular systole; such splits I believe result from a venous reflux wave produced by a sharp flapping action of the valves in the jugular bulb.

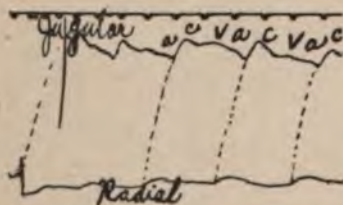


FIG. 14. — Combined venous and arterial tracing in the jugular.

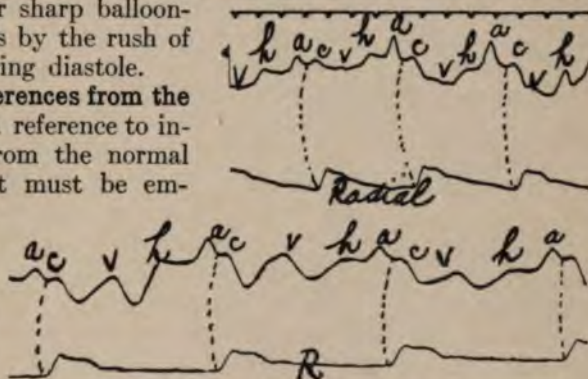
occasional blending of venous and arterial tracings; the *c-v* waves then

The *c* wave is sometimes split (Figs. 11, 12, 13). When this occurs, the first part is usually high and its fall sharp. It is due to the fling of the lever caused by placing the cup too close to the carotid, or to causes similar to those of the dicrotic notch of the radial (*q.v.*). Another variation of the phlebogram is the

resemble an arteriogram (Fig. 14). The *v* wave is sometimes divided; this division is probably caused by the two factors in its production (*q.v.*) acting somewhat asynchronously.

Another wave — the so-called *h* wave — is sometimes seen in mid-diastole or directly preceding the *a* peak (Figs. 15, 16). It is regarded as due to the rather sharp ballooning of the tricuspids by the rush of inpouring blood during diastole.

Limitation of Inferences from the Phlebogram. — With reference to information derived from the normal rhythmic tracing, it must be emphasized that, because of the mechanical limitations of the polygraph, because the waves measure volumetric changes, and because of the manner



FIGS. 15, 16. — These show an *h* wave.

of application of the venous cup, only very rarely can conclusions regarding auricular or ventricular energy be drawn. I have, for example, taken many venous tracings from patients with valvular and myo-

FIGS. 17-24. — Jugular tracings of patients with heart disease, showing variations in the heights of the waves. The variations are not distinctive of any type of valvular disease.

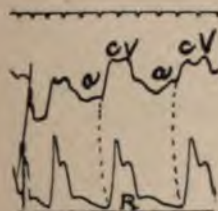


FIG. 17. — Normal phlebogram from a case of aortic regurgitation.

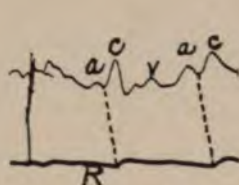


FIG. 18. — *R* = radial tracing. Normal jugular tracing from a patient with aortic stenosis and a double mitral lesion.

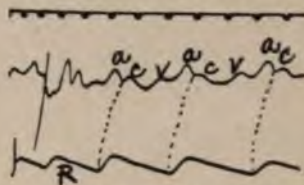


FIG. 19. — Normal jugular tracing from a patient with aortic stenosis and a double mitral lesion.

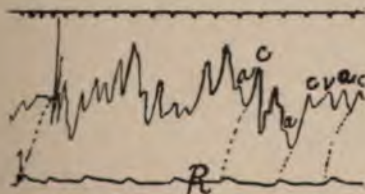


FIG. 20. — Normal sized *a* and other peaks from a case of mitral stenosis.

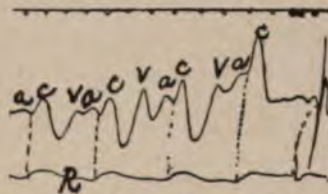
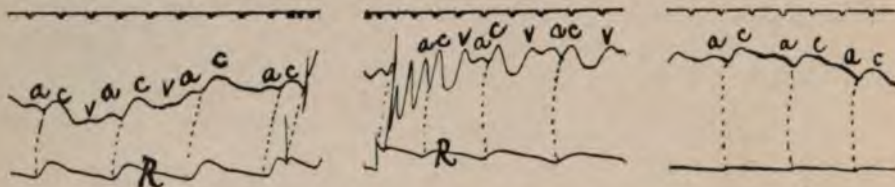


FIG. 21. — Normal *a* wave from a case of mitral regurgitation.

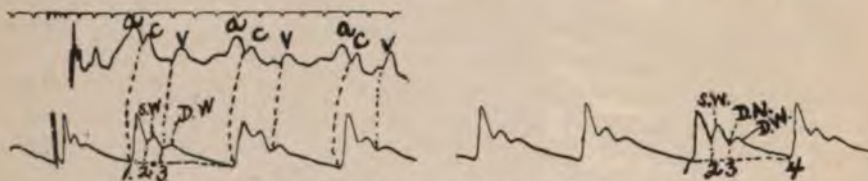
cardial lesions, and after comparison with normal tracings, I have been unable to discover any distinction between them. Figures 17-24 taken from patients with various diseases illustrate this fact.



Figs. 22, 23, 24. — Normal jugular tracing from cases of exophthalmic goiter.

The Radial Pulse. — Because of the method of instrumental application — a spring and pelotte pressing upon the radial artery — the arterial sphygmogram represents **differences of arterial pressure**. The amount of spring tension required to sufficiently occlude the artery for the purposes of tracing is quite variable, hence the resultant curve is in many instances no accurate or even approximate measure of the amount of arterial pressure. In addition to these mechanical drawbacks, extraneous factors, *e.g.* the position of the radial, its accessibility, the pliability of its walls, etc., are considerations which profoundly modify inferences drawn from the pulse tracing. The **special value** derived from the radial tracing in the polygram rests upon knowledge gained regarding **cardiac rate and rhythm**, and the aid in fixing the **time relation** of events in the cardiac cycle; the foot point of the radial serves as a standard for determining the incidence of the *c* wave in the venous tracing.

The **first wave** of the radial (Figs. 25, 26, 1-2), usually called the **primary or percussion wave**, is generally steep, its fall sharp. It is due



Figs. 25, 26. — Normal radial pulse tracings.

- 1-2 = abrupt rise probably due to instrumental fling of the lever (also called primary or percussion wave);
- 1-3 = time of ventricular systole — the aortic valves are open;
- D.N. = dicrotic notch;
- D.W. = dicrotic wave;
- S.W. = systolic wave (also called tidal and predicrotic wave).

to the sudden instrumental fling given to the pelotte and lever by the sharp impact of the onrushing blood. It occurs in, and is part of, the

wave produced by systolic arterial distention, and may be regarded as an initial artificial peak superimposed upon the arterial wave during systole. It is immediately followed by the **systolic wave**, sometimes called the **secondary tidal** or **predicrotic** wave (Figs. 25, 26, *S.W.*, 2-3). The termination of the systolic and the beginning of the dicrotic wave (Figs. 25, 26, *D.W.*) is marked by the **dicrotic notch** (Fig. 25, *D.N.*). This notch corresponds to certain events in the cardiac cycle: the end of the ventricular systole and the foot point of the *v* wave in the venous tracing (Fig. 26). Other small waves are sometimes found in the radial tracing; their cause and significance are not known.

The **cause** of the **dicrotic wave** is still in dispute. Mackenzie regards it as due to sudden relaxation of the ventricular wall, including that portion supporting the aorta: according to him there is thus developed a tendency to the production of a negative aortic wave, which is checked by the sudden stretching of the membranous aortic valves, thereby causing a second positive, the dicrotic wave. It has been experimentally demonstrated in a circulatory model in which the arterial system is represented by elastic tubing, that sudden check of the inflow produces a suction or negative pressure behind the column of fluid, and with it, resultant waves. Indeed, "dicrotic" waves have been produced in an "arterial system" in which the pumping mechanism was a syringe not comparable to the heart, a fact showing that these waves may be entirely the result of pressure effects in elastic arteries. With the influx of fluid, the tube expands; with the sudden cessation of the flow, the resultant negative pressure in a rigid tube would lead only to a reflux of fluid. In elastic tubing, however, represented in the human being by the aorta, there is the additional force of elastic recoil. Both forces—**suction** and **elastic recoil**—produce shrinkage beyond the natural caliber of the tube (aorta), the elastic constricted caliber causing secondary expansion and with it the secondary pulsatile "dicrotic" wave. Similar physical facts present in the arterial system seem to me sufficient to cause dicrotic waves. It is known, for example, that when the ventricles relax, the pressure in those chambers falls rapidly and the semilunar valves close; there is in consequence a negative pressure at the mouth of the aorta, "accompanied by an actual though slight movement of the blood current" (Wiggers). The element of elasticity in the human aorta has already been mentioned.

It has also been held that the dicrotic wave is reflected from the periphery. Under such circumstances, the distance between dicrotic and primary crests ought to diminish as the arteries recede in distance from the heart, and there should be no dicrotism in the proximal part of the compressed artery. Both of these suppositions are disproved by sphygmographic tracings. In addition the manifold arterial division at the periphery would seem to make one large reflected wave impossible.

The **height** of the dicrotic notch is ordinarily about one half of the arterial curve. In some cases, with sharp fall of arterial pressure following ventricular systole, the notch is abnormally low. This happens frequently in aortic regurgitation with cardiac failure (Fig. 27), but it

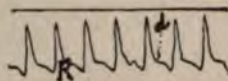


FIG. 27. — Low dicrotic notch (*d*) from a case of aortic regurgitation with decompensation.

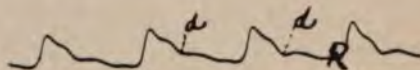


FIG. 28. — Low dicrotic notch (*d*) from a case of aortic aneurism with cardiac failure.

is also found in other decompensated heart lesions, valvular or myocardial in origin (Fig. 28).

Types of Pulse. — From what has preceded, it is evident that only exceptionally can definite conclusions regarding the “strength” or “weakness” of the circulation be drawn from graphic records. In fact, the terms “weak” and “strong” pulse are usually misapplied to what should properly be called “soft” and “hard,” respectively. The old terms previously in use have the advantage of describing the physical impression given to the examining finger. When thus descriptively applied and no deductions regarding the state of circulation are drawn, they serve a useful purpose. These terms and their definitions are as follows: a sudden fall of the pulse wave produces what is known as the “**collapsing**” pulse; if extreme, it becomes the typical “**water hammer**” or **Corrigan pulse**. The radial pulse can also be described as large and expansile (**pulsus magnus**), small or compressible (**p. mollis**), or hard and incompressible (**p. durus**). The rise of the pulse wave may be quick (**p. celer**) or slow (**p. tardus**). If dicrotism becomes palpable, the pulse is known as **dicrotic**; if the dicrotic notch breaks low and the dicrotic wave is marked, it is called **hyperdicrotic**. Occasionally, the pulse wave feels unduly sustained at the point of its maximal pulsation and falls slowly,—the **anacrotic** pulse. The **bisferiens** gives the sensation of a double pulsatile impact; it is produced by the rather equal split of the systolic plateau by the predicrotic or instrumental wave. Its assumed significance as evidence of aortic stenosis is not borne out by clinical experience.

Important information of the state of the **radial artery** is sometimes gained by careful palpation. Marked nodosity, thickening, and tortuosity are immediately apparent. The opposite information—that of a normal elastic arterial wall—is not so readily derived. If the artery is emptied by compression and the collapsed vessel palpated, the radial, if normal, is barely definable as a separate strand; if the radial is thickened and its walls stiff, the emptied artery is palpable below the point of compression.

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CHAPTER IV

THE ELECTROCARDIOGRAM

Fundamental Physiological Considerations.—The electrocardiogram is based upon the fundamental physiological fact that any muscle upon contracting produces a definite, though minute amount of electricity.

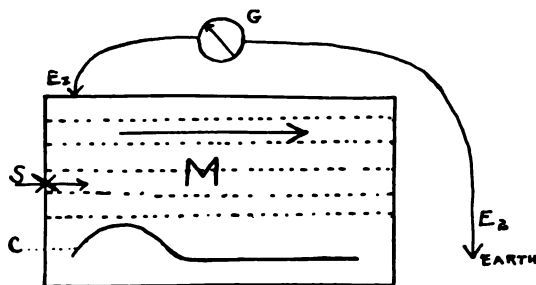


FIG. 29. — Scheme of monophasic action current. (Modified from Kraus and Nicolai — "Das Elektrokardiogramm.")

M = muscle consisting of parallel fibers;
 S = point of stimulation;
 E_1 = non-polarizable electrode;
 E_2 = earthed electrode;
 G = galvanometer;
 C = resultant monophasic curve.

The current thus produced, if allowed to pass through a sensitive galvanometer, causes deflection of the needle. For example, a muscle consisting of parallel fibers (Fig. 29, M) is stimulated at the point S , the surface at that area is connected by means of a non-polarizable electrode (E_1) to the galvanometer (G), and the other electrode (E_2) is grounded so that it remains constant (equi-

potential); then the muscle at S in contracting becomes electrically negative relative to the remainder of the musculature. As the current passes, the stimulated end becomes quiescent; the electropotential falls and reaches zero; meanwhile the needle of the galvanometer is deflected and then comes to rest. If recorded, such deflection would be a **monophasic** curve (Fig. 29).

If, instead of being grounded, the electrode is connected with the other end of a parallel fibred muscle (Fig. 30, E_2) and the latter is stimulated at the point S (Fig. 30), the stimulated area in contracting becomes electronegative, the other areas relatively positive. The current flows from $-E_1$ to $+E_2$ (Fig. 30) and produces a current

PLATE II

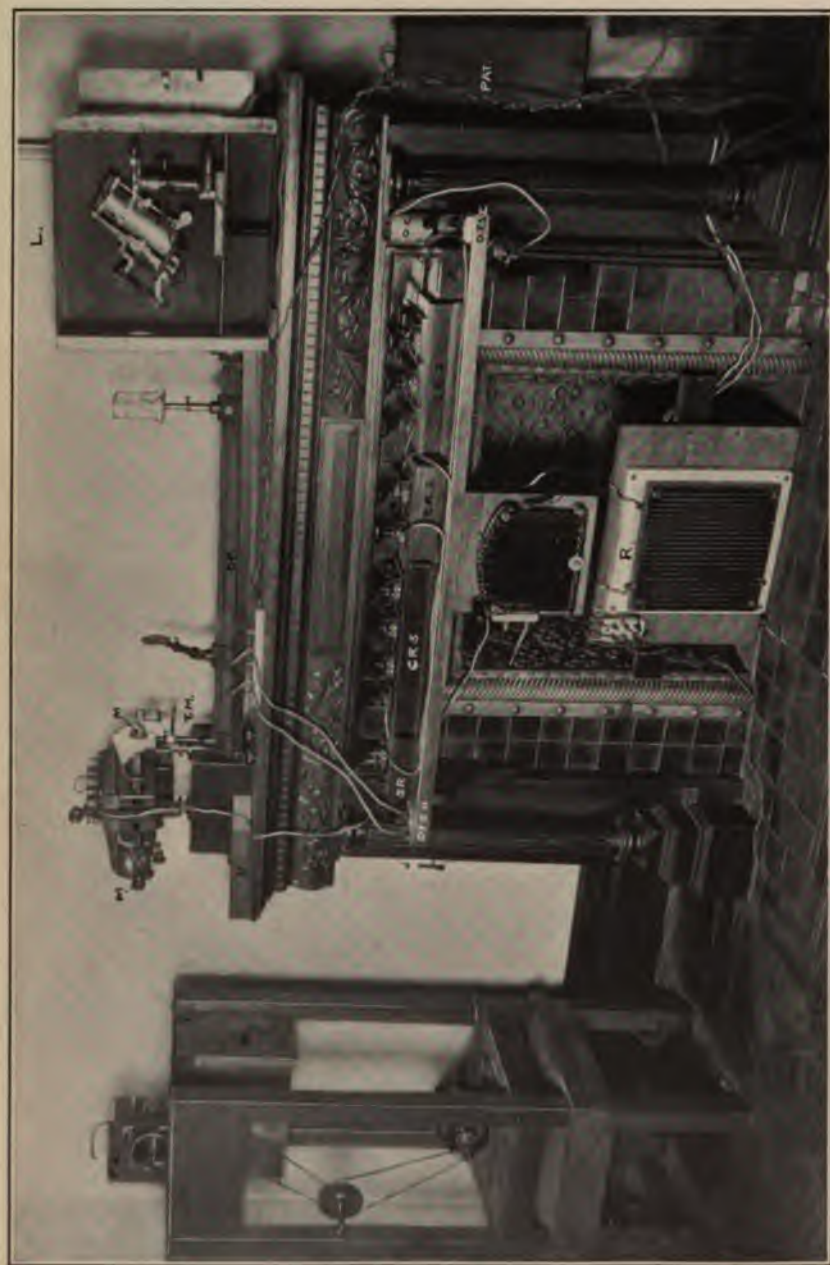


FIG. 32. — Photograph of the Electrocardiographic Apparatus in Position.¹

L. = Arc lamp;
O.P. = Optical bench;
T.M. = Time-marker;

F.H. = Box containing roll of film;
B = Box into which the finished
 film drops;

M.O. = Motor for driving film;
R = Rheostat for storage batteries;
 Other letters same as for Fig. 31.

¹ I am indebted to Dr. H. B. Williams, of the Columbia College of Physicians and Surgeons, for generous assistance and important suggestions in the installation of the galvanometer.

in the galvanometric circuit. When the contraction wave arrives at the center of the muscle, no current is induced because this zone draws negative ions in equal amounts from both sides. The two latter being positive, there is no difference in potential and the needle is not deflected. With the passage of the contraction wave toward E_2 , the latter becomes electronegative (the negative pole); the current now flows from $-E_2$ to $+E_1$ and the needle is deflected in the opposite direction. In other words, with change in electrical signs the needle is again deflected and a **diphasic curve** results.

In view of later electrocardiographic considerations, it is important to emphasize that the resultant curve depends upon the **direction** of the contraction wave in the muscle and upon the **point** at which the contraction arises. It further depends upon the **axis** of the muscle mass in relation to the electrical wave. It is also evident that the course of the contraction wave, of the galvanometric deflections, and of areas of relative negativity are intimately correlated.

These simple considerations regarding the action of a contracting muscle upon a galvanometric needle will serve to clarify the principles underlying the electrocardiographic apparatus. The latter as first constructed by Einthoven consists essentially of a fine conducting fiber lying in a narrow space between two approximated poles of a powerful electromagnet. The fiber or string is deflected by currents induced in it. The string is so sensitive and delicate that it is deflected by very weak currents drawn from the surface of the body. It is usually made of an exceedingly fine quartz fiber coated with silver. Its thickness varies from .002 mm. to .005 mm., its resistance from 1500 to 7000 ohms. The electrocardiographic apparatus which I use—a standard type—is schematically shown and described in Fig. 31. Figure 32 (Plate II) shows the apparatus set in position.

Method of taking an Electrocardiogram—the Three Leads or Derivations.—There are various methods of employing non-polarizable electrodes to conduct the potential produced by cardiac activity to the galvanometer. One method consists in wrapping flannel bandages, each about 6 inches wide and 9 feet long, thoroughly soaked in a strong warm salt solution (6 oz. of salt to one pint of water), around each forearm and around the left leg of the patient. After these extremities have

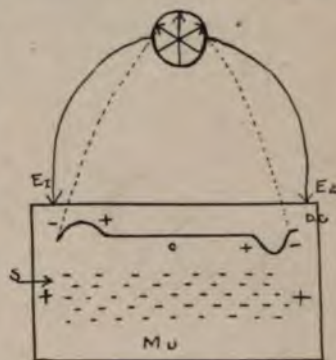


FIG. 30.—Scheme of diphasic action current. (Considerably modified from Kraus and Nikolai.)

M_u = parallel fibered muscle;
 C = center point;
 S = point of stimulation;
 E_1, E_2 = non-polarizable electrodes.
 Minus signs over M_u show that that area has become electronegative when reached by the contraction wave, and that both ends are then temporarily positive. (See text.)

been covered by a few turns, German silver electrodes with binding posts are included in the folds of the bandage. These electrodes are 6 inches long and 5 inches wide and are sufficiently thin and pliable to be bent and snugly applied. When patients can sit up, a simpler and more expeditious method consists in having three separate vessels

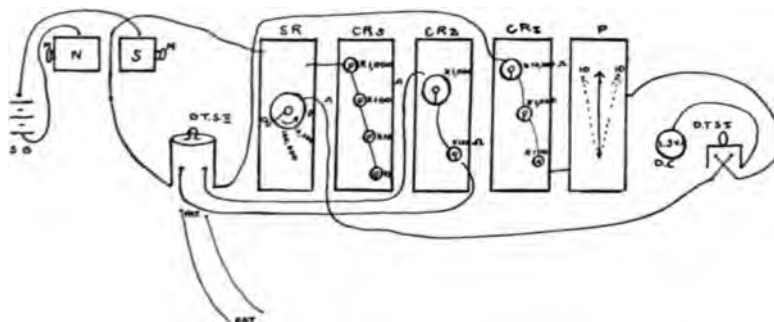


FIG. 31. — Scheme of rheostat and connection with the galvanometer.

N, S, are the North and South poles of the electromagnet activated by the storage batteries *S.B.* The latter are connected with the street current for recharging.

Between the poles of the magnet lies the silvered quartz fiber (the string), *St.*

D.T.S I and *D.T.S II* are double throw, double pole switches.

C.R 1, C.R 2, C.R 3 are crank rheostats; each crank with 11 stops (including zero) arranged in banks as figured; the ohms in each bank in multiples of 10.

S.R is a shunt rheostat.

P = potentiometer.

D.C = dry cell with a voltage of about 1.3 volts.

Each rheostat has a separate function. The resistance of *C.R 1* is thrown in so that when put in circuit with the potentiometer by switch *D.T.S I*, the former indicates 10° , a specially arranged, arbitrary figure which equals one milliampère. *C.R 1* is also in circuit with *D.T.S II* connected with the galvanometer. *C.R 2* is used to test the deflection time of the string and to measure, if necessary, the patient's resistance. *C.R 3* contains four banks. Each stop of the bank marked 2 is equivalent to one millivolt, and is used for the purpose of standardization. The shunt rheostat *S.R* is used to reduce the sensitivity of the galvanometer.

After both forearms and left leg of the patient are put into salt solution (see text and Fig. 33) they and the potentiometer are thrown into circuit by the switches *D.T.S II* and *D.T.S I* respectively. The shunt rheostat *S.R* is then moved successively upon the stops in the direction of the arrow. The string moves a variable distance from the center with each stop. It is brought back each time by throwing in resistance from the banks of *C.R 3*. Usually resistance from bank 3 is sufficient. Occasionally bank 4 is required. Finally the tension of the string is adjusted and standardized by its milled screw, so that a millivolt of current (*C.R 3*, bank 2) causes a deflection of 1 cm. The movements of the string are magnified approximately 600 times by the microscope (*M.M*) and then photographed upon a moving photographic film. In this manner tracings of various lengths can be obtained. This is an important consideration, especially when studying cardiac mechanisms which are only occasionally abnormal. A time-marker crosses the field every one-fifth second and this serves to standardize events of the cardiac cycle.

containing strong warm salt-water solutions. In each vessel is placed a porous cup containing a 100 per cent zinc sulphate solution and a zinc plate with a binding post. Thus electrical connection is established between the patient and the electrocardiographic apparatus (Fig. 33, Plate III). In either case, binding posts are connected by wires, distinguished by varying colors, and are placed in circuit with the galvanometer by means of plugs and switches. The heart, in contracting,

PLATE III



FIG. 33. — Photograph of patient with electrodes connected with the galvanometer.

gives rise to waves of electric potential which spread from their source over the entire body. It is these that are conducted to the galvanometer by means of the non-polarizable electrodes. There are thus three arbitrary directions of the current coursing through the heart, which correspond to the three extremities. These are the so-called "leads" or "derivations." The current (Fig. 34) from the right to left arm (*R.A.-L.A.*), running chiefly across the base of heart, is the first lead or derivation; that from the right arm (*R.A.*) to the left leg (*L.L.*), approximately parallel to the long axis of the heart, is the second, sometimes called the "strong" lead; that from the left arm (*L.A.*) to the left leg (*L.L.*) is the third lead and draws off the current coming mainly from the left side of the heart.



FIG. 34. — Diagram illustrating the three leads.

The photographic reproduction of the deflections of the galvanometric string during the registration of the various leads constitutes the electrocardiogram. While the electrocardiograms of no two persons are exactly alike — they may indeed be quite dissimilar — and while physiological differences vary within wide limits, there is a general conformity to

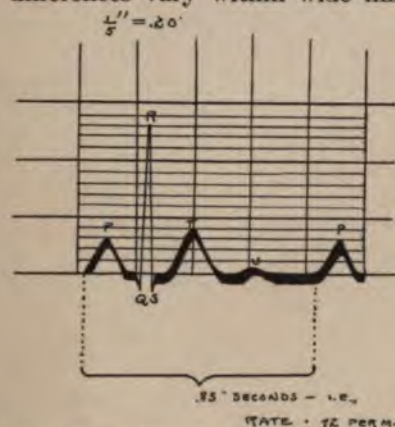


FIG. 35. — Schematic representation of a typical electrocardiogram, second lead. Each horizontal space measures one millivolt. The ordinates measure one-fifth second. The form and size of the various deviations are seen, as well as the time required for their formation.

a normal type. A typical normal electrocardiogram is schematically shown in Fig. 35, which represents the various waves or deviations, and the approximate time required for each, when the heart is beating rhythmically at the rate of 72 per minute. Each division parallel to the base line (the line of isopotential) is equivalent to one millivolt (10^{-4} volt). The electrocardiogram, that is, the registration of the difference of electric potential, precedes the actual cardiac contraction by about .03 second.

The Normal Electrocardiogram.

— The normal electrocardiogram will now be considered. The nomenclature I shall adopt is that first introduced by Einthoven and, with few exceptions, now in general use. Following the sequence of normal cardiac activity, the electrocardiogram is conveniently divided into that produced by auricular contraction, — the **auricular complex**,

—and that by ventricular contraction,—the **ventricular complex**. Animal experimentation and observations on the human subject with normal and abnormal rhythms, especially those with heart block and auricular fibrillation, have confirmed the fact that the auricular complex (known as the *P* wave) is caused by auricular contraction. This wave is directed upward, that is, it is electrically base-negative (base-active); it is usually somewhat flattened at its summit. After reaching the base line it is succeeded by a short horizontal isoelectric line, an evidence of quiescence of electric potential. The ventricular complex consists of the *QRST* deviations. The *Q* and *S* vary in size; they are usually short, sharp peaks directed downward (electrically, base-positive or what is the same thing, apex-negative or -active, “negativity” and “activity” being synonymous); they may, however, be absent. Lewis terms all downwardly directed deviations *S* waves. I have followed Einthoven’s method by which the direction of *R* is derived from the formula $R I = R II - R III$ (Chapter V). Based upon the electrophysiological facts already described, this appears to give a more rational and less arbitrary reason for the names of the different ventricular peaks. The *R* deviation, the most prominent of all the waves of the normal electrocardiogram, is directed upwards a distance varying from 10 to 15 millivolts. As measured at the base line, the time required for its formation varies from .02 to .05 second. Because of the quick deflection of the string, the *R* deviation appears as a fine line. The *T* wave slopes gradually; the down stroke is somewhat thinner than the upstroke, the summit is broad and flattened. The *R* and *T* waves are sometimes called the **first** and **second ventricular spikes**, respectively. The *U* wave rises only slightly above the isoelectric line; it is not always present. All curves are taken with a known deflection time of the string. The latter is determined by rapidly throwing in and cutting out a millivolt of current with the string tension at its usual standard (Fig. 36, Plate IV). The limit of accuracy for this deflection time is approximately .02 second; when slower, the string registers curves inaccurately flattened and low.

Variations from the Normal Type. — The usual variations are absence of the *U* wave; marked differences in the height of the *R* in the several leads; a split *R* wave with thick sides or summit; abnormally large, flat, or diphasic *T* waves, especially in leads 2 and 3; a low, flat, or split *P* wave or its absence in one of the leads; deep *Q* and *S* waves and a so-called *QRS* complex (*q.v.*). While the cause of some of these variations is known, the etiology of others is obscure or still in dispute.

The Normal Electrocardiogram and its Interpretation. — A typical normal electrocardiogram — three leads — of an adult with a normal heart is shown in Fig. 37. The *R* deviation is tallest in the second, the “strong” derivation (Fig. 34), which leads off the current in a direction parallel to the long axis of the heart. Certain basic facts regarding

the electrocardiogram are necessary to the reader in order more readily to comprehend the difference in size and direction of the deviations in the various leads in normal and abnormal hearts. The electrocardiogram is the graphic representation of the **spread of the electrical impulse** throughout the heart.

With normal cardiac rhythm, the peaks or deviations of the electrocardiogram represent at any instant of time the total differences of electrical potential. The *QRS* and probably the *T* deviations are caused by the spread to both ventricles of the excitation wave along the auriculo-ventricular junctional tissue. An upwardly directed *R* peak denotes activity of the ventricular base relative to the remainder of the

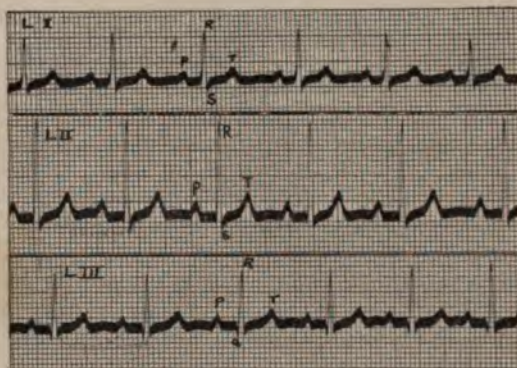


FIG. 37. — Normal electrocardiogram. *L I*, *L II*, *L III* are the three leads. The darker shaded ordinates measure one-fifth second. The horizontal spaces, one millivolt each. The *S* wave is scarcely discernible in *L III*. The *R* wave is tallest in *L II*. (Courtesy of Dr. A. E. Cohn.)

musculature. The course and the spread of the muscular contraction and of the electrical excitation waves are intimately connected, so that activity and electrical negativity are interchangeable terms. This may be proved experimentally by placing two electrodes, one upon the right ventricle in front, the other upon the apex; if the right ventricle is first stimulated, the *R* is deviated upward (base-active); if the apex, the resultant deviation is downward (apex-active).

Influences Affecting the Electrocardiogram.—From what has been said regarding monophasic and diphasic curves and their fundamental electrophysiological causes, it is evident that purely **physical considerations** affecting a contracting—and therefore an electrically excited—muscle may influence the resultant electrocardiogram. For example, let us in Fig. 30 conceive the muscle to consist of fibers of various lengths running in various directions, instead of being of the same length and parallel; we should then expect some change in the electrocardiogram from the original simpler conception. If, in addition, we imagine the muscle volume so changed that instead of an evenly cut straight muscle we are dealing with larger and smaller irregular masses, there will again be changes from the original monophasic curve. These physical considerations actually apply to the electrocardiogram derived from the human heart.

It has already been pointed out that the muscular architecture of the heart is an extremely intricate one, and that there are layers which

run in various directions from one chamber to the other (Chapter I). This phase of pure mass consideration as affecting and influencing the electrocardiogram has been admirably summed up by A. E. Cohn as due to the "disposition and volume of the muscular mass of each pair of cavities." Volumetric and mass considerations apply not only to the involved architecture of the normal heart, but also to diseased and hypertrophied hearts. For example, it seems probable that the hypertrophic process does not always, or perhaps not even regularly, express itself by hypertrophy of one chamber as compared with the other, but rather as a process affecting the fundamental complicated muscle layers.

Applying these considerations, it can now be understood how changes in muscular volume and mass profoundly influence the size and direction of the electrocardiographic deviations. It is likewise evident that any classification based upon differences of disposition and volume of the cardiac musculature will meet with numerous exceptions, the causes for which it may be impossible to fathom. Bearing these limitations in mind, the following tabulation is offered in an attempt to clarify many of the causes for variations from the normal electrocardiographic standard.

Disposition and Volume of the Ventricular Musculature as Affecting the Electrocardiogram. —

- A. Horizontally disposed (squatty) hearts.
- B. Vertically disposed (drop) hearts.
- C. Cardiac displacements.
- D. Congenital dextrocardia.
- E. Phasic variations with breathing.
- F. Ventricular hypertrophy (left and right).
- G. Ventricular dilatation (left and right).
- H. Abnormal rocking motion of the ventricle.

A. Horizontally Disposed (Squatty) Hearts. — If from any cause the heart lies abnormally flat upon the diaphragm, the ventricles are apt to be 'disposed' with a preponderant balance to the left, as diagrammatically illustrated in Fig. 38, D. In some clinical cases of squatty heart, *R III*¹ is either dwarfed or negative. Leaving the question of hypertrophy for later consideration, examples of squatty hearts are found especially in obese, middle-aged individuals. Electrocardiograms from several such patients are shown (Figs. 39, 40, 41, Plate IV). Cardiac symptoms are rarely present. Fluoroscopic examinations (Chapter IX) reveal the ventricular mass lying flat upon the diaphragm; the dome of the latter is less curved, and the diaphragmatic excursion is reduced in range. These factors are of importance in the etiology of the abnormal position of the ventricles. Gaseous distention of the stom-

¹ The numerals placed after the deviations refer to the latter in their respective leads.

PLATE IV

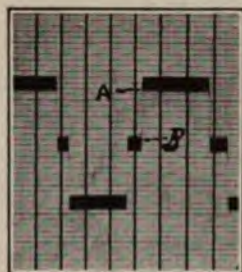


FIG. 36. — Electrocardiogram showing the deflection time of the string. The points *A* and *B* are about .02 of a second apart.

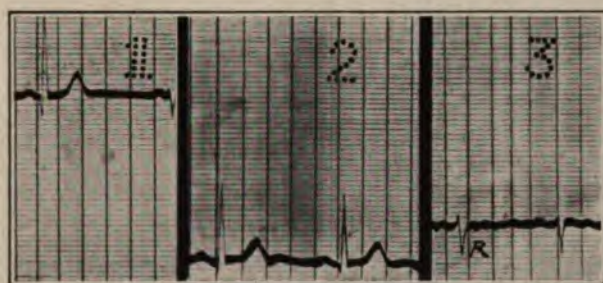


FIG. 39.



FIG. 40.

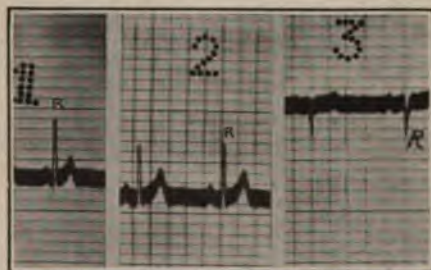


FIG. 41.

FIG. 38, 40, 41. — Electrocardiograms of patients with normal hearts and with negative *R* in *L III*. In all these cases the ventricle lay flat upon the diaphragm.

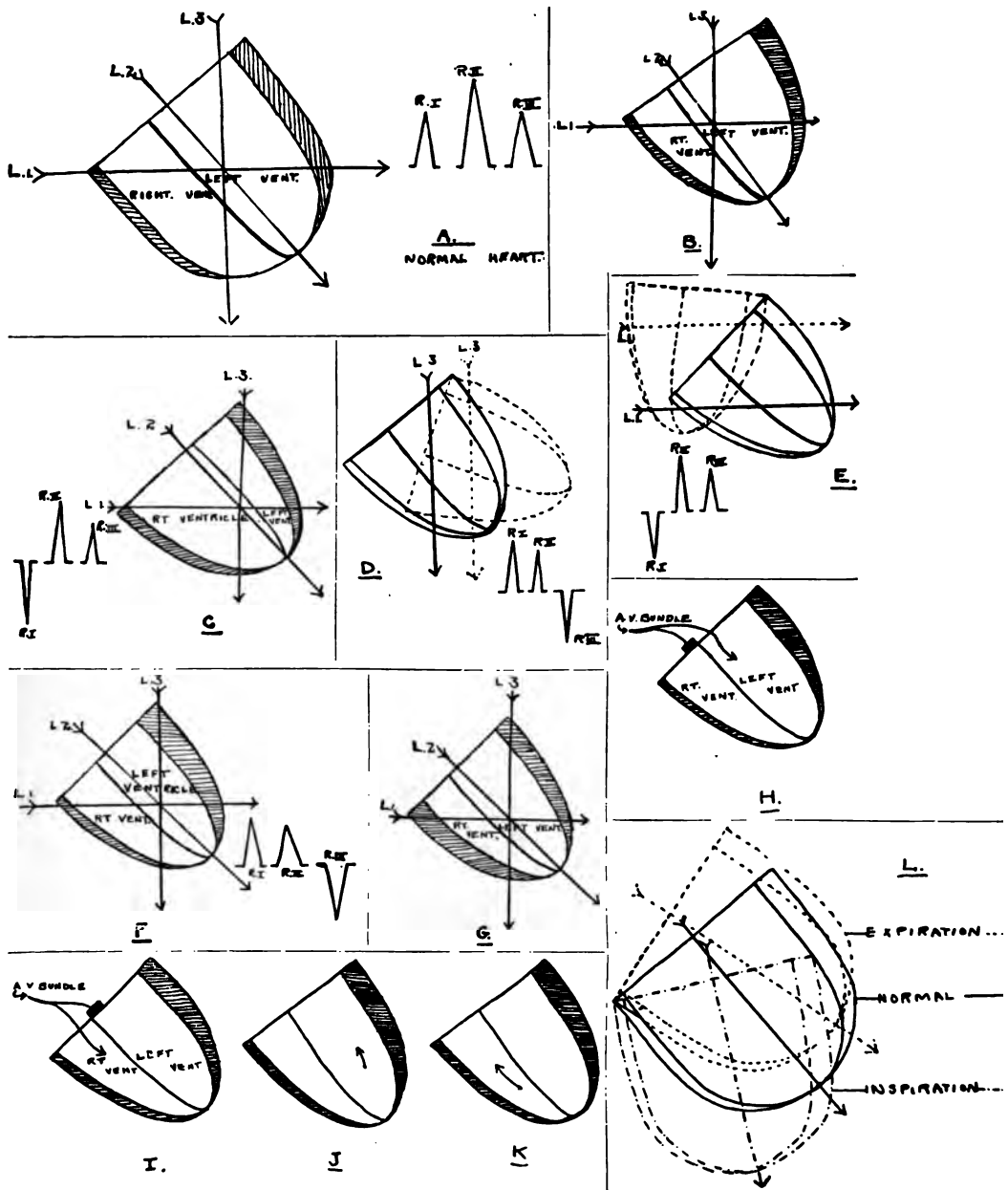


FIG. 38. — Schematic representations showing the angles made by the direction of the leads with varying ventricular axes and the corresponding electrocardiograms. Although the ventricles are schematically of different sizes, it is here assumed that the volumetric contents of both chambers are alike.

A = normal heart;
 B = left ventricular dilatation;
 C = right ventricular dilatation;
 D = left ventricular hyperbalance;
 E = right ventricular hyperbalance;
 F = left ventricular hypertrophy;
 G = right ventricular hypertrophy (electrocardiogram as in C);

H = block of right bundle branch;
 I = block of left bundle branch;
 J = left ventricular extrasystole;
 K = right ventricular extrasystole;
 L = change of position of the heart with inspiration and expiration.

ach may have a similar effect upon the position and action of the diaphragm, and hence upon the position of the heart.

B. Vertically Disposed (Drop) Hearts.—Quite opposite, theoretically, to the foregoing is the distribution of the ventricular musculature when the heart is narrow and lies vertically in the chest. The assumed muscular axis is then diagrammatically represented in Fig. 38, *E*; *RI* becomes abnormally small or negative. Clinically, patients with vertically directed (so-called "drop") hearts are apt to be young, tall, and gaunt individuals with loosely hung hearts. With the fluoroscope (Chapter IX) the entire organ appears narrow and graceful, and for the most part hidden behind the sternum. At times, the apex scarcely touches the diaphragm. An illustrative electrocardiogram is shown in Fig. 42 (Plate V).

C. Cardiac Displacements.—The heart can be displaced, as is known, by pleural exudates, adhesions, mediastinal tumors, etc. If the heart is displaced laterally so that there is no disturbance of the

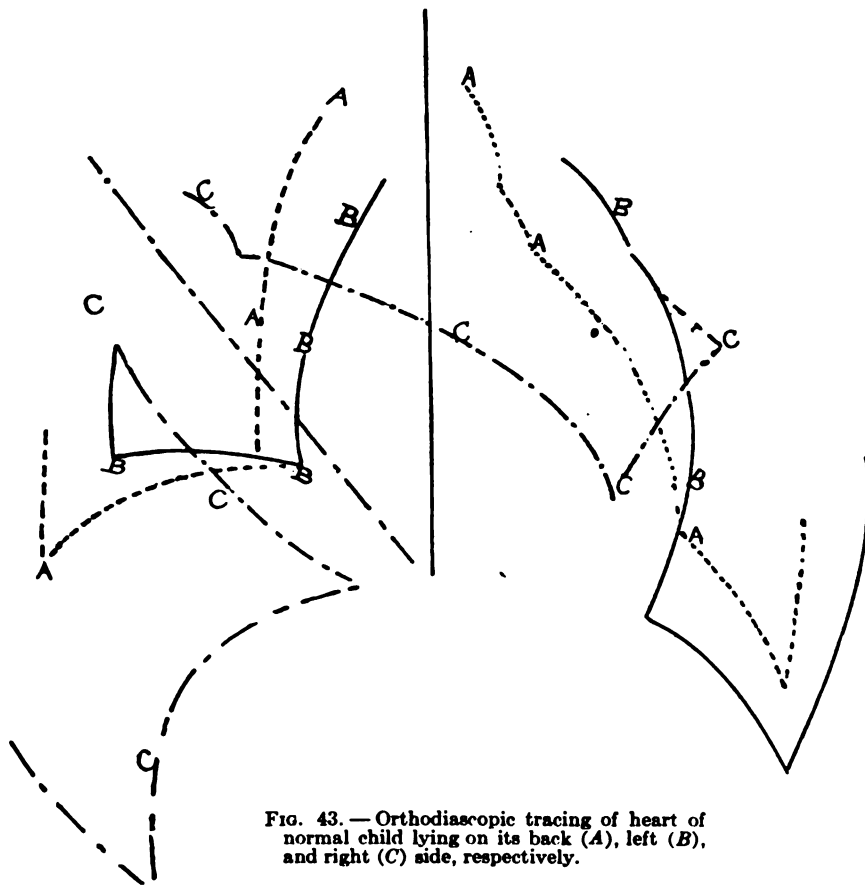


FIG. 43.—Orthodiascopic tracing of heart of normal child lying on its back (*A*), left (*B*), and right (*C*) side, respectively.

PLATE V

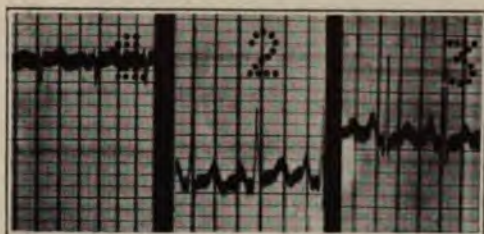


FIG. 42. — Electrocardiogram of patient with narrow heart ("drop" heart) illustrating right ventricular hyperbalance.

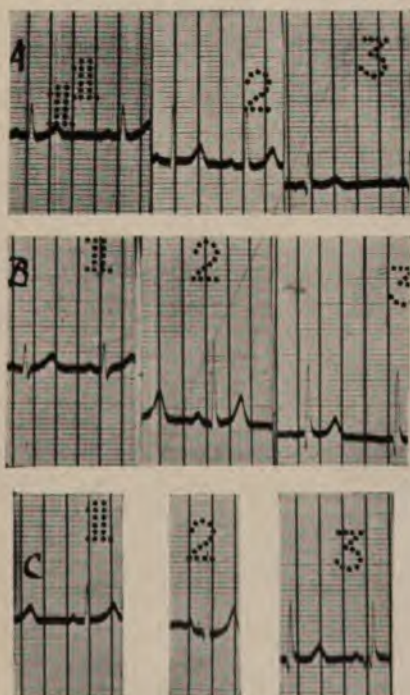


FIG. 45. — Electrocardiogram of a child whose orthodiascopic tracing is Fig. 148. The letters A, B, and C again refer to the position of the child on its back, left and right sides respectively.

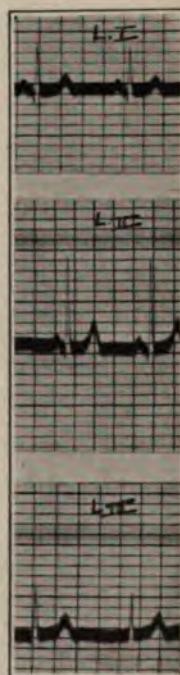


FIG. 47. — Acquired dextrocardia with normally directed deviations in all three leads.

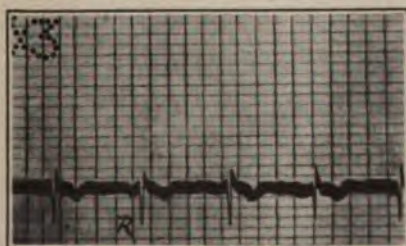


FIG. 48. — Slight phasic variation with breathing.

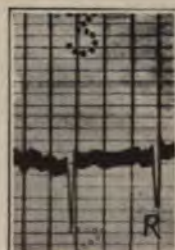


FIG. 49. — L II. Slight phasic variation with breathing.

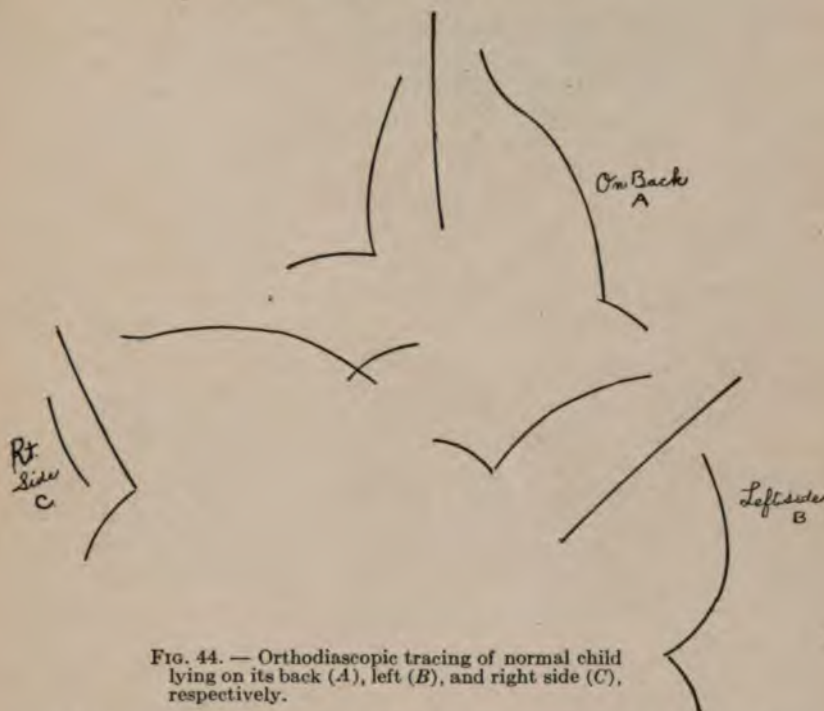


FIG. 44. — Orthodiascopic tracing of normal child lying on its back (*A*), left (*B*), and right side (*C*), respectively.

muscular balance of the heart, the electrocardiogram does not change. The latter is only affected when the changes in the cardiac position cause a change of the plane of electrical potential in relation to the fixed body planes (represented by the "leads"). The effect of displacement can be best exemplified in children, and is illustrated in Fig. 43, *A, B, C*, which are the orthodiascopic tracings of a healthy boy of ten lying alternately on his back, left and right sides. The tracings show marked variations in the disposition and contour of the heart, mainly due to ventricular rotation and consequent foreshortening or lengthening of the cardiac outline. Another illustration of a mobile heart is Fig. 44 (positions *A, B, C*), orthodiascopic tracings of the normal heart of a child of seven lying alternately on her back, left and right sides. Corresponding electrocardiograms were taken (Fig. 45, Plate V, *A, B, C*). In addition to slight changes in the *Q* and *S* deviations, the heights of the *R* in the *A, B, C* positions varied as follows: Lead *I*, $R = 6^A, 5^B, 6^C$ (the numbers refer to the number of millivolts of deflection); in Lead *II*, $R = 10^A, 15^B, 16^C$; in Lead *III*, $R = 6^A, 13^B, 10^C$.

D. Congenital Dextrocardia. — As a corollary to the observations already made regarding the effect of change of the planes of electrical potential upon the electrocardiogram, it is apparent that congenital dextrocardia in the first, sometimes called the symmetrical lead, will

produce electrocardiographic deviations exactly opposite in direction to the normal (Fig. 46). All the peaks are directed downwards instead of upwards; *R III* becomes taller than *R II*. Such an electrocardiogram offers indubitable proof of congenital dextrocardia and serves to distinguish the latter from acquired right-sided malpositions of the heart due to fluid in the chest, pulmonary tumors, pneumothorax, adhesions, etc. For example, Fig. 47 (Plate V) is the electrocardiogram of a patient with an acquired dextrocardia, in whom the heart was drawn into the right chest by adhesions following a right-sided pulmonary abscess and subsequent pleural fistula following operation. Fluoroscopically, the heart was seen to occupy an area in the right chest prac-

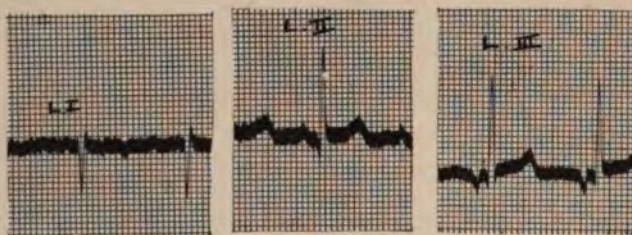


FIG. 46. — Congenital dextrocardia. All the deviations are directed downwards, in *L I*. *R III* is taller than *R II*.

tically identical with that of congenital dextrocardia. Since the heart had been pulled in a **lateral** direction only, the electrocardiographic deviations remained normal in direction.

E. Phasic Variations with Breathing. — As the result of breathing, some electrocardiograms, usually in the second and third leads, present a rhythmic waxing and waning, an increase and decrease in the size of the *R* waves. During fluoroscopy, I have often noted a marked variation in the position of the heart during respiration (Fig. 38, *L*); it is sometimes sufficiently pronounced to produce a movement of the apical portion of the ventricle through an arc of several centimeters. This shift is most evident in younger individuals with thin chest walls and large respiratory excursions of the diaphragm; it is least in patients with fat abdominal walls and broad hearts. The heart moves with the base as a comparatively fixed point. During inspiration there is a descent, in a clockwise direction, of the ventricular mass, especially of its apical portion. The heart tends to assume an erect position. The rise of the diaphragm during expiration produces a contrary effect; the left ventricle then moves anti-clockwise. These movements, when extreme, necessarily affect the muscular disposition of the ventricles and alter the electrocardiogram. This is usually most noticeable in the third lead, probably because the left ventricle is especially influenced by the respiratory phases. *R III* becomes taller during inspiration and smaller during expiration as the ventricular mass tends to assume a more vertical or horizontal position, respectively (Fig. 38, *L*).

PLATE VI

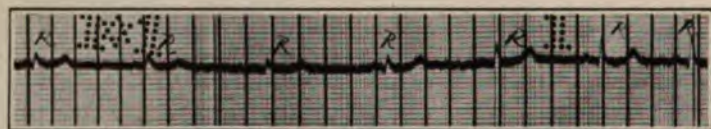


FIG. 50. — Marked phasic variation with breathing.

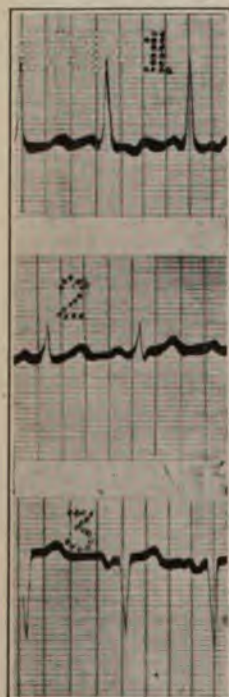


FIG. 51. — Aneurismal dilatation of the aorta with left ventricular hypertrophy. Negative *R* III.

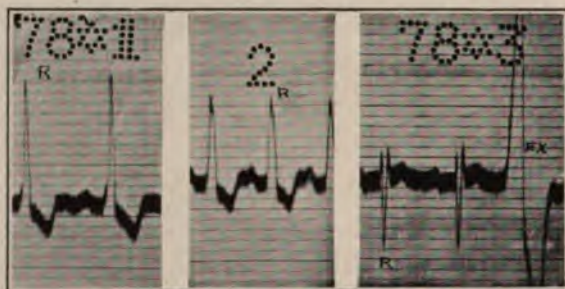


FIG. 52. — Aortic stenosis. Left ventricular hypertrophy. Negative *R* in *L* III. Ventricular extrasystole (*Ex*) in lead III.

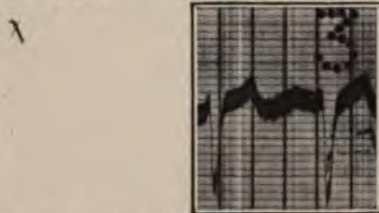


FIG. 53. — Negative ventricular deviation III. From a case of left ventricular hypertrophy and aortic aneurism.

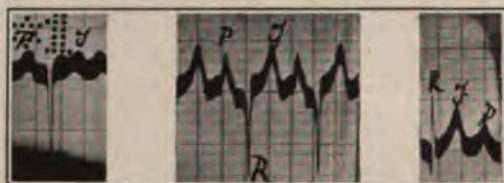


FIG. 55. — Electrocardiogram of a boy of 17 with congenital ductus arteriosus. Note negative *R* I, *R* II, evidence of marked right ventricular hyperbalance.

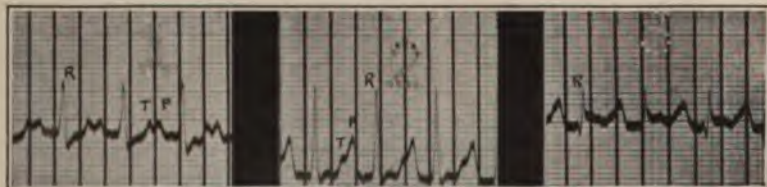


FIG. 54. — Electrocardiogram of a patient with aortic regurgitation and left ventricular hypertrophy; the *R* deviation is positive in *L* III.

Respiratory phasic variations are also found in patients with ventricular hypertrophy, but the cardiac excursion being ordinarily less, the phasic electrocardiographic variations became correspondingly limited. Figures 48 and 49 (Plate V) are examples of a moderate respiratory



FIG. 48.—*L III*. Phasic variation with breathing. Note the difference in size of the *R* deviations.

effect upon the size of the deviations. Figure 48 is the electrocardiogram of a stout individual with a normal heart lying in a squatty position; Fig. 49, from a patient with left ventricular hypertrophy. When *R III* is small, phasic variations may not only dwarf it but cause its final disappearance, as shown in Fig. 50 (Plate VI).

F. Ventricular Hypertrophy — Left and Right (Fig. 38, F, G). — It has sometimes been assumed that negative *R I* and positive *R III*, and positive *R I* and negative *R III*, are always indicative respectively of right and left ventricular hypertrophy. This observation has been partly substantiated by necropsies. On the other hand, exceptions have been noted both at post mortem and as the result of clinical examination. Some undoubted cases of left ventricular hypertrophy, for example, do not give the deviations assumed for this condition. In addition, it is by no means infrequent to find patients with perfectly normal hearts, resting normally "disposed" in the chest, who present the deviations supposedly typical of hypertrophy. It will thus be seen that there are many drawbacks and exceptions to definite conclusions regarding the existence of hypertrophy based upon the direction of the electrocardiographic deviations. As a general rule, however, it will be found that in aortic disease with evident left ventricular preponderance, *R I* is positive and *R III*, negative. Examples are given in Figs. 51 and 52 (Plate VI) and in Fig. 53. Occasionally, however, *R III* is positive, even when left ventricular hypertrophy is present (Fig. 54, Plate VI).

The electrocardiographic evidence of **right ventricular hypertrophy** is best exemplified by congenital cardiac disease due to malformation of the pulmonary artery. This is illustrated by the case of a boy of 17 with patent ductus arteriosus (Fig. 55, Plate VI). Both *R I* and *R II* are negative. Upon the theory of mass imbalance affecting the electrocardiogram, it would seem that together, negative *R I* and *R II* are indicative of very marked right ventricular hypertrophy. The same theoretical consideration applies to negative *R II* and *R III* as indicating extreme left ventricular hypertrophy, as exemplified in Fig. 56 (Plate VII), taken from a patient with aortitis, and clinical and fluoroscopic evidence of extreme left ventricular hypertrophy. Infants and young children are also apt to have the electrocardiographic complex of right

ventricular enlargement, because in them the walls of the right ventricle are relatively thick.

Cases of **mitral stenosis** do not always yield electrocardiograms indicative of right ventricular hypertrophy, a condition usually associated with this lesion. Thus, Figs. 57 and 58 are from cases of marked and typical stenoses. In Fig. 57, the main ventricular deviation is negative. The *R* deviations in Fig. 58 are positive. Whether the latter and similar apparently atypical electrocardiograms in cases in whom, clinically, right ventricular hypertrophy is assumed, are due to some of the factors already discussed (abnormally disposed ventricles, ventricular dilatations or hypertrophy) or whether they may even be due to counterbalancing left ventricular hypertrophy, it is at present impossible to state.

G. Ventricular Dilatation (Fig. 38, B, C).—Though hearts which are organically sound may occasionally become dilated as the result of

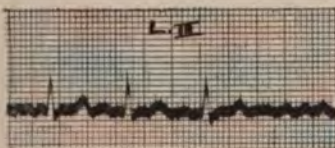
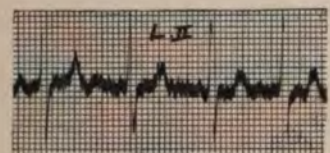
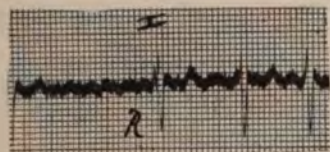


FIG. 57.—Advanced mitral stenosis. *R I* is negative. Auricular fibrillation is present.

overstrain or of tachycardial attacks, the term ventricular dilatation is here meant to apply to decompensated, diseased hearts. To a great extent, our clinical knowledge regarding cardiac dilatations, especially of one chamber as contrasted to the other, or of dilatations as differentiated from moderate hypertrophy, is meager. There exists, however, some definite evidence that cardiac dilatation following acute endocarditis is not uncommon in children. This subject has never been studied from the electrocardiographic aspect, but, as with considerations regarding the effect of mass changes, it seems probable that **muscular redistribution** resulting from dilatation can also bring about changes in the electrocardiographic deviations.

II. Abnormal Rocking Motion of the Ventricle.—During the course of routine fluoroscopic examination of normal and abnormal hearts, I have encountered occasional cases in which there was a sort

of to-and-fro, rocking motion of the ventricular mass, with the base of the heart acting as the fixed area. At times the entire mass, at others only its apical portion, was seemingly involved in this abnormal motion. It is possible that this abnormal motility, whatever its cause, when sufficiently marked to disturb muscular mass relations, may well produce a change in the electrocardiogram. Its mechanism may be compared to that causing phasic variations with breathing (*q.v.*). In the latter, however, the motion is comparatively gentle and slow and

PLATE VII

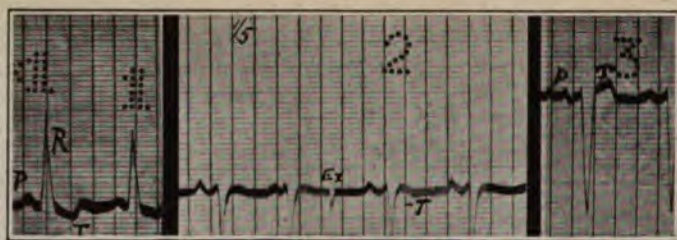


FIG. 56. — Electrocardiogram showing an interpolated extrasystole (*L II, Ex*). *R* is abnormally wide, especially in *L I* and *L III*. The direction of the *R* deviations in the various leads indicates marked left ventricular hypertrophy. *T I* and *T II* are negative, presumed evidence of myocarditis. (From a patient with left ventricular hypertrophy and luteic aneurismal dilatation of the entire thoracic aorta.)

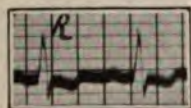


FIG. 59. — Electrocardiogram showing somewhat thickened *R* summit.



FIG. 60. — Electrocardiogram (*L II*) showing slightly notched *R* summit.

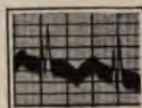
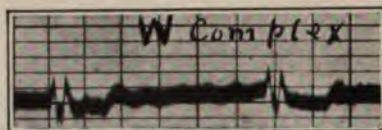
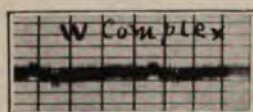


FIG. 61. — Electrocardiogram showing split *R* (*M* complex).



FIGS. 62, 63. — Electrocardiograms showing split *R* (*W* complexes).

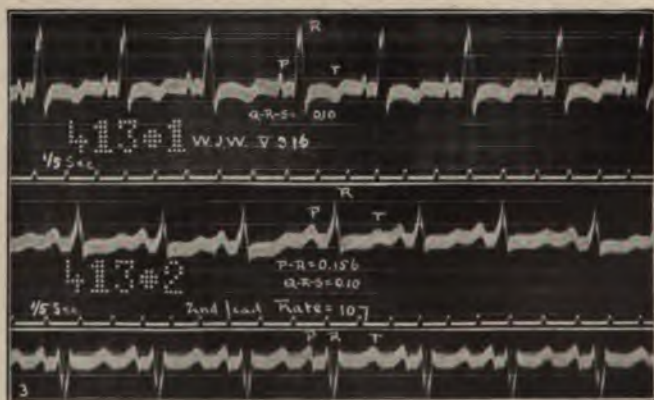


FIG. 65. — Electrocardiogram showing intraventricular block. (Courtesy of G. C. Robinson: *Archives of Internal Medicine*, 1916, XVIII, 845.) Note the notched and splintered complexes.

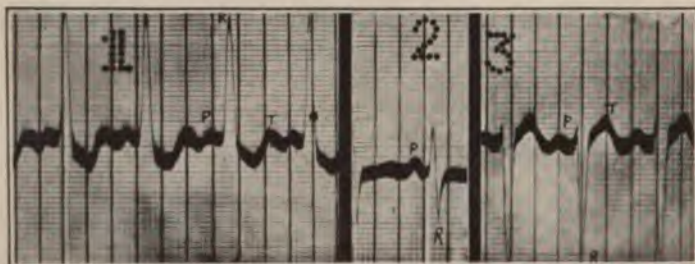


FIG. 66. — Electrocardiogram showing ventricular complexes of abnormal width, but of normal form.

is not followed by a second rocking motion, which is characteristic of the to-and-fro motion alluded to.

The possible effects of this type of abnormal motility will be mentioned in connection with the *M* and *W* complexes (*q.v.*).

Asynchronous Auricular and Asynchronous Ventricular Activity — Split, Splintered, and Notched *R* and *P* Deviations, and *M* and *W* Complexes. — In addition to variations in the electrocardiogram due to disposition and volume of the ventricular musculature, in which physiological synchronous activity of the chambers has been assumed, there are conditions in which, for various reasons, there is a **retardation** of the **excitation wave** in one chamber as compared with its fellow. This is seemingly responsible, in the main, for many of the "split" and "notched" complexes.

The normal difference in contraction time between the two ventricles may amount to as much as .03 second. If there is a slight retardation of the excitation waves in the ventricles, due to abnormal ventricular

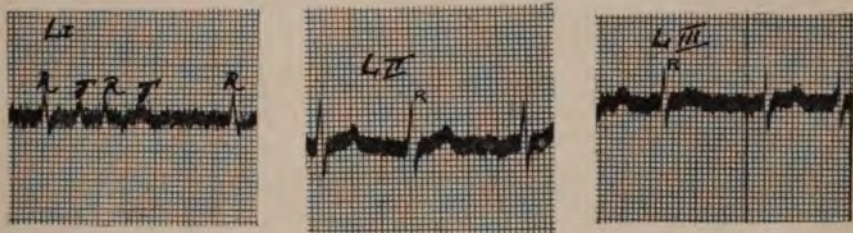


FIG. 58. — Advanced mitral stenosis with auricular fibrillation. All the *R* deviations are positive.

asynchronism, it may account for the thickening of the *R* deviation at its summit (Fig. 59, Plate VII). It may also account for various degrees of notching at the apex (Fig. 60, Plate VII). If the notching is extreme, there is a sharp division of the *R* peak into two similar components. When the latter are directed upwards, the complex resembles the letter *M*. The letter *M* is an appropriate term, I believe, for this composite wave (Fig. 61, Plate VII). When the components are directed downwards, the complex resembles the letter *W* (Figs. 62, 63, Plate VIII).

Some of the complexes which I have termed *M* and *W* have been called the *QRS* complex by Lewis. Because of the similarity of their components, many of these seem composed of two separate *R* deviations with or without *Q* and *S* deflections. The *M* and *W* complex may be assumed to be due to **asynchronous ventricular contractions** if the outlines of the waves are sharp, and if when measured at their base lines, they consume not more than twice the time required for the normal *R* deviation; that is, a basic width of the *M* or *W* of about .06 of a second. (See Intraventricular Block.) Even when the individual deviations are small, the resemblance to the letters *M* and *W* is sufficiently accurate to warrant their descriptive use, especially when qualified as "dwarfed,"

"flat," or "low." These letters may thus serve to distinguish and separate many of the electrocardiograms found in the heterogeneous and somewhat confused *QRS* groups. While many of the *M* and *W* complexes are probably due to ventricular asynchronism, it seems possible that the to-and-fro ventricular motion already described (*q.v.*) may also be productive of similar complexes; or even that a sharp twist of the ventricles during contraction may disturb ventricular mass relations and produce a deep *Q* or *S* wave. I have observed fluoroscopically several instances of ventricular rocking motion accompanied by *M* or *W* complexes, or deep *Q* and *S* waves.

Similar in etiological significance to the notched *R* are the notched or split *P* deviations (Fig. 64) which occasionally give rise to the appearance of two distinct undulations.

Such complexes are probably due to asynchronous auricular contractions. A notched *P* is found most often in mitral stenosis.

Intraventricular Block.—

Recently, Oppenheimer and Rothschild have described a *QRS* complex in which the time required for the completion of this group was prolonged beyond 0.1 second, approximately the normal limit. The *R* wave was abnormally broad; instead of clean and sharp sides, it was slightly or considerably notched and broken. The waves were usually of low amplitude in

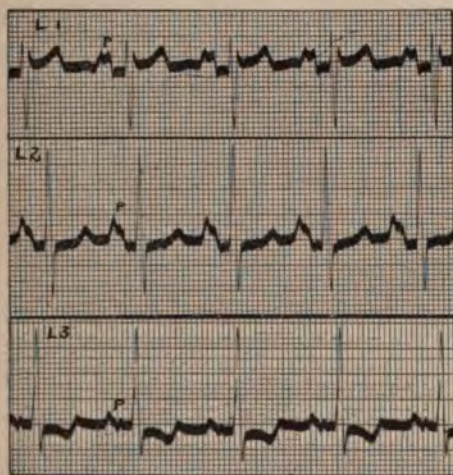


FIG. 64. — Notched *P* wave from a case of mitral stenosis. (Courtesy of Dr. A. E. Cohn.)

all leads. These observers found this electrocardiographic complex in cases in which there was marked arteriosclerotic or cardiovascular-renal disease, usually associated with severe myocarditis. In four cases they were able to corroborate the clinical picture by necropsy examination; this showed sclerosis especially in the endocardial and subendocardial layers; that is, in the neighborhood of the terminal arborizations of the conduction system. They believe the abnormal electrocardiogram is the result of intraventricular block, thus interfering with the normal and orderly spread of the electrical excitation wave throughout the heart. It should be pointed out, however, that hearts presenting the above clinical or pathological picture need not necessarily produce the abnormal electrocardiogram described.

G. S. Robinson has also reported a series of cases whose *QRS* complexes required .10 or longer for their completion (Fig. 65, Plate VII).

PLATE VIII

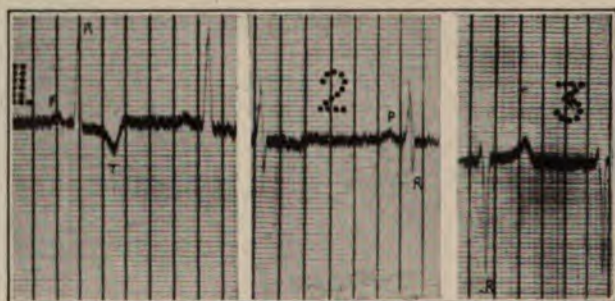


FIG. 67.

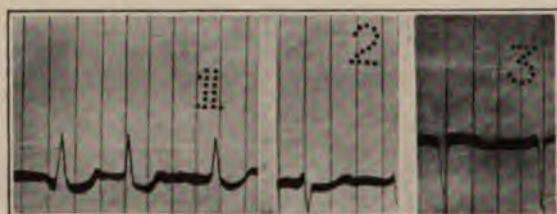


FIG. 68.

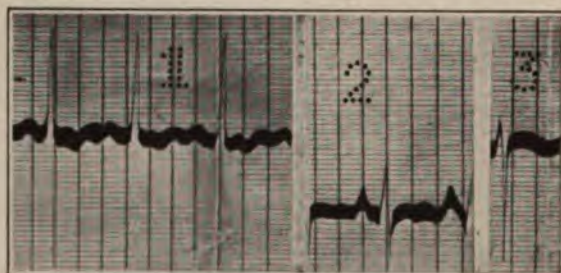


FIG. 69.

FIGS. 67, 68, 69. — Electrocardiograms showing ventricular complexes of abnormal width but of normal form. *R* is not abnormally wide in all leads.



FIG. 73. — Electrocardiogram showing tall *T II*.



FIG. 74. — Electrocardiogram showing *T* taller than *R*.

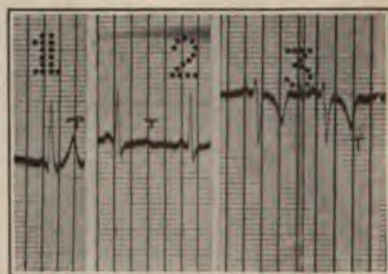


FIG. 75. — Negative *T III*. From a case of aortic stenosis with left ventricular hypertrophy.

These were likewise assumed to be due to derangement of intraventricular conduction from hindrance to the excitation wave, either along the normal paths or at the normal rate. In most of the cases from whom the electrocardiograms were taken there was clinical evidence of a profound disturbance in the muscular efficiency of the heart.

The "Wide R."—Both of the above types of intraventricular block are characterized, as has been stated, by a broken and notched complex comprising the *QRS* group. In an entirely different category belong those cases that I have studied, in whom the *R* deviations are **normal in shape and form**, whose sides are unbroken and not notched, and in whom there was no evidence of a bundle branch lesion. The characteristic of my cases was the **abnormal length of time** required for the completion of the main ventricular wave. The normal time for the completion of the latter varies from .02 to .05 second. I have adopted as a standard a width of .07 or over as being an "abnormally wide *R*." A few cases with their electrocardiograms are herewith epitomized: Fig. 56 (Plate VII) is from a patient who had general anasarca from luetic cardiosclerosis. *R I* = .12 second, *R II* = .07, *R III* = .13. Figure 66 (Plate VII) is from a case of dilatation of the arch of the aorta and moderate left ventricular hypertrophy; the clinical diagnosis was cardiosclerosis. *R I* = .12, *R III* = .08. Figure 67 (Plate VIII) is from a physician of 53 who developed scarlatinal nephritis at the age of 15. At present the urine contains albumen and casts. The patient has the physical signs and symptoms of moderate cardio-nephritis. *R I* and *R II* each equals .07. Figure 68 (Plate VIII) is from a woman of 68 who, when first seen, was suffering from anasarca from cardiovascular-renal disease. Under proper therapy, she improved remarkably. *R I* = .09. Figure 69 (Plate VIII) is from a male patient of 55 suffering from hypertension and moderate cardiosclerosis. *R I* = .07. The electrocardiograms of all these patients were taken during the stage of compensation.

In all, I have observed 18 cases which presented an *R* width of .07 of a second or over. All of these cases showed unmistakable signs of severe cardiac disease, and at one time or another were decompensated. Clinically, most of the cases had hypertension and left ventricular hypertrophy. Myocardial insufficiency alone was not the cause of the abnormally wide complex, for most of the electrocardiograms were taken when symptoms of decompensation were slight or absent. Ventricular dilatation, another possible assumption for the abnormally wide *R*, can also probably be disregarded for the same reason. Besides, in a series of decompensated valvular cases that I observed in whom ventricular dilatation was a marked feature, the *R* complexes were of normal width.

Although the fundamental cause of the wide *R* in my cases is not apparent, it is probably due either to **delay** in the **development** of **electrical excitation** or to **delay** in its **propagation**. Since the complexes are normal in form, the excitation wave has apparently followed a

normal path in the ventricles. Delayed propagation seems the more probable factor. As in intraventricular block, severe cardiosclerotic disease is assumed to cause the notched and delayed *QRS* complex, a wide *R* of normal shape but of abnormal width may conceivably be due to patches of myocardial thickening scattered throughout the ventricular wall in amounts sufficient to impede and abnormally prolong the excitation wave.

It should be mentioned that I have studied the complexes of other cardiovascular cases who were clinically as ill as the "wide *R*" cases but who presented no abnormally broad complexes. The reason for this I have not been able to discover.

Of the 18 cases studied, the *R* complex was rarely abnormally broad in all leads. Since the first, second, and third leads draw off the cardiac current from various directions,—namely, breadthwise, diagonally, and lengthwise, respectively,—one may at least hypothesize that the diseased myocardium lay chiefly in one cardiac plane, thus producing a wide *R* in the corresponding lead. In a general way, those cases which clinically showed the most myocardial disease were the ones in whom the *R* complex was widest.

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CHAPTER V

MATHEMATICAL CONSIDERATIONS UNDERLYING THE ELECTROCARDIOGRAM

In previous chapters, the fact was emphasized that the electrocardiographic deviation represented the resultant of the differences of electrical potential existing at any given moment. The diagnostic significance of the direction of the *R* wave was also discussed. The elementary mathematical principles, upon which differences in the *R* and other deviations depend, require brief comment.

Stress has been laid upon the fact that the size of the *R* peaks depends upon the angle formed by the "leads" and the "electrical axis"; the nearer the electrical axes approach parallelism to the leads, the taller the corresponding peaks; the nearer these approach a right angle, the smaller the peaks. The size of the deviation thus varies with the angle made by the lead and electric axis. In other words, the electrocardiogram is modified in each lead by the relation of the direction of the current to that of the lead. Conversely, the angles made by these two lines may be computed from the difference in size of the deviations in the three leads.

Manifest Size. — Einthoven has differentiated the **actual** size of the deviations, as reproduced in the electrocardiogram, from their "**manifest**" size or the "**manifest**" difference of electric potential. He defines the latter as that dimension in millivolts which is derived when the electrical axis and leads coincide; it is the **maximal** size of the electrical potential of the heart in that lead. The manifest size of any deviation is computed from the registered actual electrocardiogram. Einthoven and his pupils, Fahr and de Waart, have laid down mathematical principles by which it is possible to estimate the angles formed by the electrical axes with the leads. If, for purposes of simplification, the lines of the three leads be conceived as forming the sides of an equilateral triangle (Fig. 70, *R, L, F*) and its middle point, *H*, represent the heart, the size of the *R* deviations in the three leads is obtained by the right-angled projection of the deviations upon the electrical axis passing through the point *H*.

From these, data are derived for the determination of the angle formed by the electrical axis and the leads.¹

Measurement of the Angles Made by the Leads and the Electrical Axis. — If smaller angles be discarded and, for purposes of clinical approximation, only those considered which are multiples of 30°, the computation of $E1:E2:E3$ (Fig. 70) is much simplified; for example,

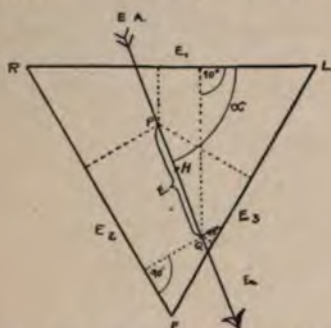


FIG. 70. — (From Einthoven, Fahr, and De Waart — "Ueber die Richtung und Manifest Grösse der Potentialschwankungen," etc. *Archiv f. d. ges. Physiologie*, 1913, CL, 308). The right and left arms and left foot (designated R, L, F , respectively) form the angles of an equilateral triangle. The point H is in the center and represents the heart. If the arrow EA represent any given electrical axis, the angle it forms with the first lead (RL) be represented by α , and any given length pq be called E , the right angle projection of its length will give its corresponding value in the various leads; that is, in LI it will equal $E1$; and LII , $E2$ and in $LIII$, $E3$. The distances $E1, E2, E3$ are proportional, that is, $E1:E2:E3$. Since any angle of an equilateral triangle is equal to 60°, the following trigonometrical formulæ are derived:

$$\begin{aligned} E1 &= E \cos \alpha; \\ E2 &= E \cos \alpha - 60^\circ; \\ E3 &= E \cos (120 - \alpha); \\ E3 &= E2 - E1. \end{aligned}$$

The formula $E3 = E2 - E1$ is of special importance, for, given the heights of deviations in any two leads, the height of the remaining peak may be derived.

When

$\alpha = 0^\circ$, then $E1:E2:E3 = 1:+.5:-.5$

$\alpha = 30^\circ$, then $E1:E2:E3 = 1:1:0$

$\alpha = 60^\circ$, then $E1:E2:E3 = +.5:1:-.5$

$\alpha = 90^\circ$, then $E1:E2:E3 = 0:1:1$

etc.

From these numerical proportions, the angle α can be approximated within 30°. The "manifest" equals the actual size of $R1$ ($E1$, Fig. 70) when α equals 0°; of $R2$ ($E2$) when α equals 60°, and of $R3$ ($E3$) when it equals 120°. For the exact determination of this angle, tables are required. In the Appendix to this chapter (*q.v.*) there is one table for approximation within 10°; by the use of the second table (Interpolation Table) the exact angle may be computed. For example, if $R1$ equals 3.2, $R2$ equals 12.5, and $R3$ equals 9.3, then $E1$ ($R1$): $E2$ ($R2$): $E3$ ($R3$) = 3.2 : 12.5 : 9.3. To derive an approximation within 10° (Table I), 12.5, the tallest deviation, becomes the denominator, thus: $\frac{10}{12.5}$.

Substituting this fraction for 12.5 in the equation, we find $E1:E2:E3 = 2.6:10:7.4$. Furthermore, from the same table we note these figures determine that the angle α falls between 70° and 80°; and that $E1$ between 70° and 80°

¹ (See also legend with Fig. 70.) To insure mathematical accuracy it is necessary to measure the R deviations at identical phases of the heart cycle. With this in view, two galvanometers may be simultaneously employed; one to record sound records, the other to record the electrocardiograms. The position of the Q, R and S in identical cardiac phases is thus determined. Although this method is necessary for absolute accuracy, unless phasic differences of the heart cycle produce marked changes in the electrocardiogram, measurements derived from the ordinary electrocardiogram are sufficiently exact for clinical purposes.

has a value between 3.5 and 1.8, a difference of 2.7. In Table II (Interpolation Table, Third Column) the nearest approximation to this difference is 2.6, which is equivalent to 6° ; hence, $\alpha = 70^\circ + 6^\circ = 76^\circ$.

As another example, suppose $T 1 = 4$, $T 2 = 1.5$, and $T 3 = -3.5$, we then have the proportion $E 1 : E 2 : E 3 = 4 : 1.5 : -2.5$. To derive approximation within 10° we multiply by the fraction $\frac{10}{4}$, 4 being the tallest deviation. Then $E 1 : E 2 : E 3 = 10 : 3.75 : -6.25$. From Table I, this proportion shows that the angle α is between 0° and -10° , and also that $E 2$ varies from 5 to 3.5. In Table II, its nearest approximation is 3.8, an angle of 8° . Hence, $\alpha = 0^\circ - 8^\circ = -8^\circ$.

Since the value of the angle alpha can be computed, it is possible by trigonometry¹ to determine the manifest size (Fig. 70) of the various deviations.

In a schematic diagram by Pardée (Fig. 71), based upon the Einthoven conception of the leads forming a triangle with the heart in the



FIG. 71. — (From H. E. Pardée — "Form of the Electrocardiogram," Journal of the American Medical Association, 1914, LXII, 1311). a , b , c , represent the numerical value of the deflection caused by an action current in leads I, II, III, respectively. $b = a + c$ and will be directed upwards or downwards according as a and c are directed; + before the letter signifies an upward deflection; - signifies a downward deflection.

center, the angle α can be roughly measured by conceiving the direction of the electrocardiographic current as lying in one of 6 sectors of 60° each. Thus, currents or electrical axes between $+30^\circ$ and $+90^\circ$ (the normal segment) give $+R 1 + R 2 + R 3$.

¹ The formulæ by which the value of E (the manifest size) may be derived are :

$$E = \frac{E 1}{\cos \alpha}$$

$$E = \frac{E 2}{\cos(\alpha - 60^\circ)}$$

$$E = \frac{E 3}{\cos(120^\circ - \alpha)}$$

Axes between $+90^\circ$ and $+150^\circ$ give $-R_1 + R_2 + R_3$

Axes between $+150^\circ$ and -150° give $-R_1 - R_2 + R_3$

Axes between -150° and -90° give $-R_1 - R_2 - R_3$

Axes between -90° and -30° give $+R_1 - R_2 - R_3$

Axes between -30° and $+30^\circ$ give $+R_1 + R_2 - R_3$

These results are diagrammatically shown in Fig. 72.

By these computations and methods, accurate knowledge of the direction and size of the electrical axis and of the electrical balance of the heart is obtained, but definite information regarding the **origin** of the excitation wave is not thus derived. A knowledge of the manifest size in conjunction with the electrical axis helps to diagnose the most



FIG. 72. — Diagram showing the direction of the leads with currents (electrical axes) at various angles. The circle is divided into six sectors of 60° each. The direction of the currents is marked by the arrow heads, the ventricular deviations for the respective leads by R_1 , R_2 , R_3 .

likely point or points of origin of the excitation wave. By further amplification and applications of the Einthoven formulæ, Fahr and Weber have concluded as follows: The deviation Q results from excitation in the neighborhood of the middle zone of the heart: the papillary muscles and their environment. The apex of R is the resultant of excitation in the base of the heart, usually over its middle portion. This area is ordinarily to the left, but sometimes to the right of the middle line. The deviation S in general denotes apical negativity; the electrical center is usually to the left, but may be to the right of the median line. The deviation T is the end of the excitation wave. As a rule, it results from excitation of the ventricular base, to the right or left of the median line.

It is thus apparent that the electrical excitation wave in the ventricle normally proceeds in the following sequence: the neighborhood of the papillary muscles, the base of the heart, the apical portion, and finally the basal area.

APPENDIX ¹

CHAPTER V

TABLE I

TABLE II

α	REGISTERED POTENTIAL DIFFERENCES e			MANIFEST POTENTIAL DIFFERENCES E	INTERPOLATION TABLE			
	e^1	e^2	e^3		Differences in Degrees	Potential Differences + e	Differences in Degrees	E
					0°	0	10°	$11,5$
0°	10	5,0	-5,0	10,0	I {	2° 0,4	8°	11,3
10°	10	6,5	-3,5	10,2		4° 0,8	6°	11,1
20°	10	8,2	-1,8	10,7		6° 1,2	4°	11,0
30°	10	10	0	11,5		8° 1,5	2°	10,8
40°	8,2	10	1,8	10,7		10° 1,8	0°	10,7
50°	6,5	10	3,5	10,2	II {	2° 2,2	8°	10,5
60°	5,0	10	5,0	10,0		4° 2,5	6°	10,4
70°	3,5	10	6,5	10,2		6° 2,9	4°	10,3
80°	1,8	10	8,2	10,7		8° 3,2	2°	10,2
90°	0	10	10	11,5		10° 3,5	0°	10,2
100°	-1,8	8,2	10	10,7	III {	2° 3,8	8°	10,1
110°	-3,5	6,5	10	10,2		4° 4,1	6°	10,1
120°	-5,0	5,0	10	10,0		6° 4,4	4°	10,0
130°	-6,5	3,5	10	10,2		8° 4,7	2°	10,0
140°	-8,2	1,8	10	10,7		10° 5,0	0°	10,0
150°	-10	0	10	11,5				
160°	-10	-1,8	8,2	10,7				
170°	-10	-3,5	6,5	10,2				
+180°	-10	-5,0	5,0	10,0				
-170°	-10	-6,5	3,5	10,2				
-160°	-10	-8,2	1,8	10,7				
-150°	-10	-10	0	11,5				
-140°	-8,2	-10	-1,8	10,7				
-130°	-6,5	-10	-3,5	10,2				
-120°	-5,0	-10	-5,0	10,0				
-110°	-3,5	-10	-6,5	10,2				
-100°	-1,8	-10	-8,2	10,7				
-90°	0	-10	-10	11,5				
-80°	1,8	-8,2	-10	10,7				
-70°	3,5	-6,5	-10	10,2				
-60°	5,0	-5,0	-10	10,0				
-50°	6,5	-3,5	-10	10,2				
-40°	8,2	-1,8	-10	10,7				
-30°	10	0	-10	11,5				
-20°	10	1,8	-8,2	10,7				
-10°	10	3,5	-6,5	10,2				
0°	10	5,0	-5,0	10,0				

¹These tables and the examples in the text were taken from Einthoven, Fahr and de Waart "Ueber die Richtungen und die Manifest Grösse der Potentialschwankungen, . . . des Elektrokardiogramms." (Arch. f. d. ges. Physiologie, 913, *cf.* p. 275.)

CHAPTER VI

COURSE OF THE EXCITATION WAVE

By means of careful experimental investigations based upon measurements of electrocardiograms led off from different parts of the cardiac surface, Lewis clearly demonstrated that the excitation wave in the auricle spreads, ripple-like, from the sino-auricular node as a center; it follows the chief auricular muscle bands which radiate from this region, and spreads thus throughout the auricular tissue and septum to reach the *A-V* bundle. "The excitation wave appears in some parts of the right auricle before some portions of the left, and vice-versa" (Lewis).

The spread of the ventricular excitation wave does not follow the anatomical arrangement of the musculature. After reaching the *A-V* node, it begins almost simultaneously in different parts of an area supplied most directly by the right branch of the *A-V* bundle; this is the part of the right ventricular wall which overlies the large anterior papillary muscle; later, the remainder of the right ventricle becomes active. In the left ventricle the wave also follows a definite course. The earliest point to be excited is the vortex of the left ventricle or the extreme apex; about $\frac{1}{100}$ second later the neighboring points are activated; excitation of the remainder of the left ventricle is almost simultaneous. The cardiac base is usually last affected, and its activity is practically coincident with activity in the conus region. Since some ventricular areas are excited simultaneously, the ventricular wall must be reached by simultaneous impulses traveling along a large number of paths; by experiments, these have been shown to be the Purkinje fibers. But some parts of the ventricular musculature not supplied by free, branching Purkinje strands (*e.g.* the conus region beneath the pulmonary valves) are activated very early. This is accounted for by the thinness of the ventricular musculature in the region of the right papillary muscle. The varying thickness of the musculature also accounts for excitation reaching the thinnest area first and later the thicker left ventricular vortex. Excitation is found to travel extremely rapidly through the conduction system. In brief, then, the excitation wave appears almost simultaneously along the interior of both ventricles. The activation of the ventricular surface is only partly dependent upon

its distance from the Purkinje fibers; it is mainly controlled by the muscle thickness overlying the latter, for the excitation wave is conceived as appearing on the surface by directly piercing the musculature. Thus, by a different method, a result similar to that of Fahr and Weber has been reached regarding the course of the ventricular excitation wave.

(4) VARIATIONS OF THE *T* WAVE

(See Classification, Chapter V.)

The *T* deviation is normally monophasic and directed positively (*i.e.* upwards) in the three leads (Fig. 37), its height varying between 2 and 3 millivolts. It is occasionally quite tall (Fig. 73, Plate VIII) and sometimes exceeds in height the corresponding ventricular spike (Fig. 74, Plate VIII). Not infrequently, it is deviated downwards in the third lead (Fig. 75, Plate VIII). The clinical conditions in which the *T* wave is diphasic or deviated downwards in the first or second leads are only imperfectly understood; hence, inferences drawn therefrom are for the present only tentative.

A positively deviated *T* denotes that the excitation wave has entered the ventricular base (Chapter V). In a number of cases of heart disease, especially of the myocardium, *T I*, *T II*, or both, may be small or negative (Fig. 56, Plate VII; Fig. 76, Plate IX), indications of an abnormal termination of the excitation wave in the ventricle. Much diagnostic significance has been given to these negative *T* deviations, especially since Einthoven regarded a well-marked positive *T* as a sign of good cardiac contractility, and its absence, or diminution, the reverse. It is, however, now known that the *T* wave may be absent in normal, and be very well marked and positive in diseased, hearts.

Effect of Digitalis on the *T* Wave.—It has recently been demonstrated that digitalization of a patient with heart disease may convert a positive *T* to a flattened or negative wave. Though this may be partly due to muscular ventricular redistribution as the result of relief of decompensation, the chief factor is probably alteration in the contractility of the heart muscle. Pharmacological experiments in frogs and mammals have shown that digitalis produces diminished diastolic relaxation of the apical region. In the toxic stage there is systolic standstill of the apex, while the base relaxes in diastole. It is possible, therefore, that similar influences affect the digitalized human heart, producing continued apical activity and thus a negatively deviated *T* wave. The usual effect of digitalis administration on the *T* wave may not become evident until atropine has been injected. Atropine apparently unmasks and removes the inhibitory effect of the drug; thereafter the full influence of digitalis on the heart can be observed. Some years ago, Einthoven made the observation in one case that as the result of exercise, a negatively deviated *T III* in a normal heart became positive. It has also been recently demonstrated in a few cases that a negative *T* in decomp-

pensated hearts may become positive with restoration of compensation. The change is probably the result of **nerve influence** occurring during exercise. This view is based upon the fact that exercise is accompanied by **accelerator** excitation, and that various experimental observations have shown a definite correlation between accelerator stimulation and the size of the *T* wave. It therefore seems probable that a positive *T* produced by exercise, or by the restoration of compensation, is due to a neurogenic influence acting upon the ventricular base. Whether, under these conditions, a positive *T* is to be regarded as a favorable change is an open question and awaits further corroboration. Hence, clinical inference respecting its value should for the present be guarded.

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CHAPTER VII

THE ARRHYTHMIAS — THEIR POLYGRAPHIC, ELECTROCARDIOGRAPHIC AND CLINICAL RECOGNITION

THE passage of the normal impulse, as exemplified in polygraphic and electrocardiographic tracings of the normal rhythmic cardiac mechanism, has already been described. We shall now treat of those abnormal mechanisms known as cardiac arrhythmias and irregularities. A. E. Cohn has succinctly grouped the arrhythmias as coming under variations of a few fundamental normal functions. He states that cardiac irregularities arise from the **abnormal passage** of the impulse, from **abnormal sequence of contraction** of the pairs of chambers, and from **abnormal coördination** of the muscle mass. So far as possible, these basic considerations have been incorporated into my Tabulation of the Arrhythmias. As will be seen, I have grouped almost all the arrhythmias as due to those arising in the **auricle**, those arising in the **ventricle**, and those arising in the **specialized tissues**, the **sino-auricular** and the **auriculo-ventricular nodes**.

TABULATION OF TYPES OF ARRHYTHMIAS AND CARDIAC IRREGULARITIES

- | | | |
|---|---|--|
| A. AURICULAR
ARRHYTHMIAS | { | I. Auricular Extrasystoles { from the <i>normal</i> site.
from an <i>abnormal</i> site (ec-
topic).
II. Paroxysmal Tachycardia of <i>Auricular</i> Origin.
III. Auricular Incoördination. { (1) Auricular Fibrillation.
(2) Auricular Flutter.
(3) Incoördination interme-
diate between Flutter
and Fibrillation. |
| A'. NODAL EXTRASYSTOLES. | | |
| B. VENTRIC-
ULAR
ARRHYTH-
MIAS | { | I. Ventricular Extrasystoles (from Right or Left Ventricle).
II. Interpolated Extrasystoles.
III. Automatic Ventricular Activity-Ventricular Escape.
IV. Paroxysmal Tachycardia of <i>Ventricular</i> Origin.
V. Ventricular Incoördination { (1) Ventricular Fibrillation.
(2) Branch-bundle Lesions. |
| C. TRUE BRADYCARDIA. | | |

D. ARRHYTHMIAS PRODUCED BY ABNOR- MAL SE- QUENCE OF CONTRAC- TION OF AU- RICLES AND VENTRICLES	I. Disturbance in the Sino- auricular Node	(1) Sinus Arrhythmia or Irregularity.
		(2) Sino-auricular Block (Sinus Block).
		(3) Blocked Auricular Beat.
	II. Disturbance in Atrio-ven- tricular Node	(1) Prolonged Conduction Time.
		(2) Shortened Conduction Time.
		(3) Backward Conduction.
		(4) Auriculo-ventricular Heart Block
		(a) Incomplete Heart Block.
		(b) Complete Heart Block (Dissociation).

It is the aim of graphic methods to exactly transcribe the cardiac mechanisms, normal and abnormal. It has always been the aim of progressive clinical medicine to adopt and adapt the knowledge gained by the use of exact instrumental methods for immediate use at the bedside, where instruments may not be available. It is thus that, in the light of knowledge gained by a careful study of the graphic methods, and with a full appreciation of the physiological pathology involved, it is possible to diagnose most types of arrhythmia by ordinary methods of examination. For this purpose, the stethoscope should be placed over the cardiac apex and the fingers kept on the pulse; the neck should be carefully scrutinized for jugular and carotid pulsations (*a* and *c* waves). The value of keen observation will be discussed in connection with the clinical recognition of the arrhythmias.

A I. AURICULAR EXTRASYSTOLES

Some preliminary observations regarding the general nature of extrasystoles — auricular, ventricular, and nodal — are required before their various types and instrumental and clinical recognition are discussed.

Contractions of auricle or ventricle which anticipate the normal rhythmic time of their occurrence and disturb the normal rhythm are known as **extrasystoles** or **premature contractions**. Fundamentally, the normal passage of the impulse is disturbed. Extrasystoles were formerly termed 'pulsus bigeminus.' The term 'premature contraction' is preferable because it so aptly describes the phenomenon. Extrasystole is in a sense a misnomer, for the contraction is not 'extra' or additional; it is simply anticipatory. However, since 'extrasystole' is commonly used, it will be here employed. If extrasystoles recur at regular intervals after each normal beat, the resultant rhythm is known as **coupling**, **coupled rhythm**, or **coupled beats**.

There are certain characteristics, as Lewis has pointed out, which differentiate the premature from the normal contraction. The **physiological** or **homogenetic beat** is one of a series of similar rhythmic contrac-

tions; the contractions are equally spaced; there is an orderly building up of impulse formation which requires at least one half second for each beat. This power of rhythmic impulse formation resides chiefly, if not entirely, in the specialized tissues of the sino-auricular node, of the atrio-ventricular node, or in the bundle of His (Fig. 77). Regarding the **premature, extrasystolic, heterogenetic, or pathological contraction**, whether single or multiple, there is an exceedingly rapid, abrupt, or no impulse formation. Ventricular extrasystoles, for example, may follow each other at intervals as short as .25 second or less. Extrasystoles are further differentiated from normal beats by their prematurity and by their lack of rhythmic tendency; even when multiple or occurring in



FIG. 77.



FIG. 78.



FIG. 79.



FIG. 80.



FIG. 81.

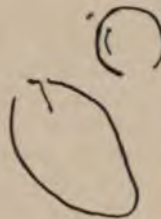


FIG. 82.

FIGS. 77-82.—Diagrams showing the origin of physiological and pathological contractions.

FIGS. 77-78.—S-A = sino-auricular node;

A-V = part of the atrio-ventricular conduction system.

FIG. 77.—XX shows the origin of homogenetic beat in the sinus region with normal propagation in the junctional tissue.

FIG. 78.—X = ventricular extrasystole — heterogeneous ectopic beat.

FIG. 79.—X = auricular extrasystole — heterogeneous ectopic beat.

FIG. 80.—X = nodal extrasystole.

FIG. 81 shows the disturbance in rhythm with a premature auricular contraction.

FIG. 82 shows the disturbance in rhythm with a premature ventricular contraction.

showers, the rate of production is usually maximal. There is no relation between the heightened activity of the physiological heart rhythm and the prevalence of extrasystoles. Often, influences which depress the one favor the occurrence of the other; such an example is chloride of potassium in the experimental animal. Premature contractions bear a different

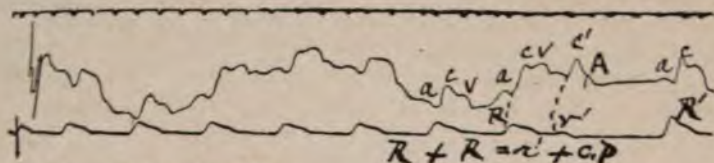


FIG. 83.¹ R = rhythmic beat;
 r' = extrasystole;

$C.P.$ = compensatory pause;
 R' = larger post extrasystolic beat.

In the jugular tracing, the premature wave c' is synchronous with r' . Its foot-point is determined by measuring off with dividers the distance Rr' from the preceding rhythmic c ($Rr' = cc'$). The auricular beat A is not premature. It falls at its rhythmic time; hence, the inter-auricular distance $a-A'$ is equal to the normal beat (R). A occurs before the c' wave has completed its fall, hence the abnormal width of the combined $c'A$. The A wave is often not indicated as a distinct part of the $c'A$ because it may be lost in the more prominent c' wave.

relation to the cardiac nerves than the normal beats; for instance, gradually increased right vagus stimulation retards the normal rhythm; it has no effect on, or produces abrupt cessation in, a series of premature contractions. It can be electrocardiographically shown that the latter

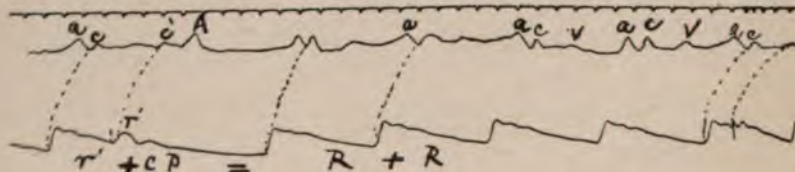


FIG. 84.

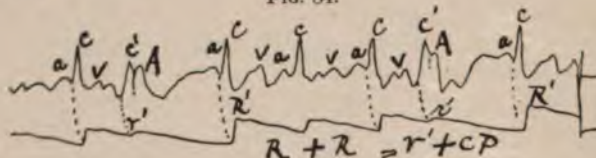


FIG. 85.

FIGS. 84, 85. — Ventricular extrasystoles with typical compensatory pauses.

rarely originate from the normal rhythmic center. Finally, the nearer the physiological beat originates in, and approaches the vicinity of, the superior vena cava, the faster the rhythm. This law does not apply to extrasystoles; their rate does not depend upon their origin in auricle or ventricle.

Premature beats (Figs. 78-82), because of their **inherent weakness** or because the wave is not **properly directed** against the aortal cusps,

¹ In all the polygraphic tracings, the time-marker measures $\frac{1}{2}$ second.

are occasionally 'frustrane' or 'abortive'; that is, they either do not open the aortal cusps or do so too slightly to produce a palpable radial wave. These frustrane contractions are sometimes termed 'missed beats,' an evident misnomer, because ventricular contractions do actually occur, but the blood is not propagated as a pulse wave. Not only may abortive contractions escape palpation, they may even be too minute to be seen in radial tracings. In such instances the phlebogram shows the usual evidence of premature contractions (Figs. 86, c' , 87 c'); the method of seeking the point of incidence of the rhythmic auricular beat has already been shown (A wave, Figs. 83, 84, 85).

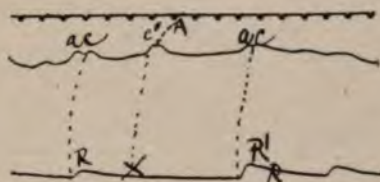


FIG. 86.

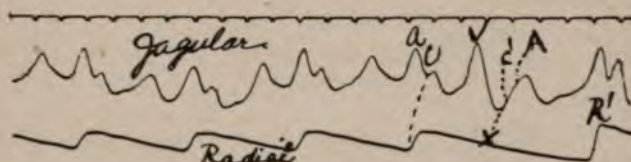


FIG. 87.

FIGS. 86, 87. — Ventricular extrasystoles with c' in the jugular but no representation in the radial tracing. X marks the point where the extrasystole should have produced a pulse wave.

Heterogenetic beats, single or multiple, that originate outside of the sinus region have been called "ectopic" contractions (Lewis). Auricular extrasystoles are usually ectopic, but this can only be demonstrated electrocardiographically by the **difference in the form** of the abnormal auricular complex (P waves, Chapter IV). "Ectopic" auricular contractions, that is, those arising from an abnormal site, are not necessarily premature in their occurrence and hence may not disturb the normal rhythm.

When auricular extrasystoles are present, the ventricle usually responds to the premature auricular contraction after normal conduction time.

As may be seen in the **polygram**, auricular extrasystoles produce radial waves of varying size and strength (r' , Figs. 88, 89); frustrane contractions are comparatively rare. The auricular extrasystole in the jugular tracing is usually marked by normal conduction time between it and the answering beat (a' , c' , Figs. 88-89); the conduction time, how-

ever, is occasionally diminished (a' , Figs. 90-91), or that of the succeeding beat, increased (Fig. 92 a). Extrasystoles are sometimes multiple

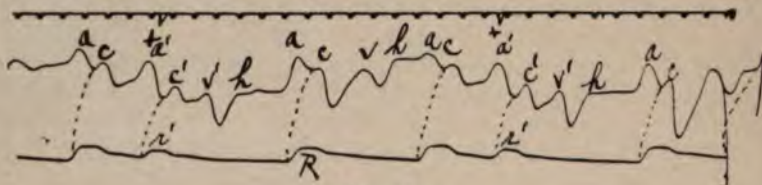


FIG. 88.

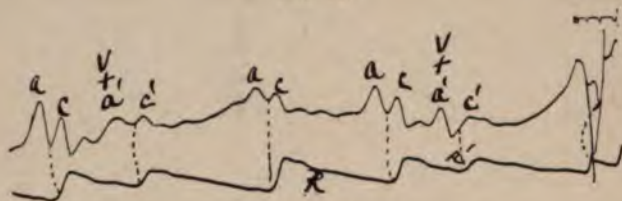


FIG. 89.

FIGS. 88, 89. — Auricular extrasystoles. The auricular beat a' is premature and is followed by the c wave. The v wave of the rhythmic beat has fallen with a' .

(Figs. 93-94). If the first one is registered in the radial curve, the latter resembles coupled rhythm; the jugular tracing will then reveal the frustrane extrasystoles (Figs. 93, a'' , c'' , 94, a'' , c'' , a''' , c''').

As seen in the **electrocardiogram**, when auricular extrasystoles originate in the **pacemaking area**, *i.e.* in the **normal** site, the complexes are identical in shape with those of the normal; the only distinguishing feature is their prematurity. The ventricle usually responds after a normal P - R interval, and its complex is the same as the other rhythmic beats because it follows the normal path in the conduction system (Figs. 95, 96, 97, Plate IX). The premature auricular contraction often falls at a time when its complex is superimposed upon the T wave (Fig. 98, Plate IX), so that upon superficial examination the P wave appears absent. Careful scrutiny, however, indicates that the composite P - T wave is somewhat taller and thicker than the P wave alone (Fig. 97, Plate IX). **Ectopic** auricular contractions which originate in an **abnormal site** outside the sinus area show their origin by change of complex. This is well exemplified in Fig. 99 (Plate X), which demonstrates beats starting from different ectopic foci (A , Ex. 1; A , Ex. 2); the abnormal complexes are deviated upwards and resemble the normal. This probably indicates an origin close to the pacemaker. It is to be noted that the ectopic beats are neither premature nor are they followed by compensatory pauses; the only feature differentiating them from the normal is their difference in form.

Clinical Recognition of Extrasystoles (Premature Contractions). — Isolated extrasystoles which are followed by compensatory pauses are

PLATE IX



FIG. 76. — Electrocardiogram showing diphasic $T I$, negative $T II$ and $T III$. From a patient with hypertension and myocarditis.

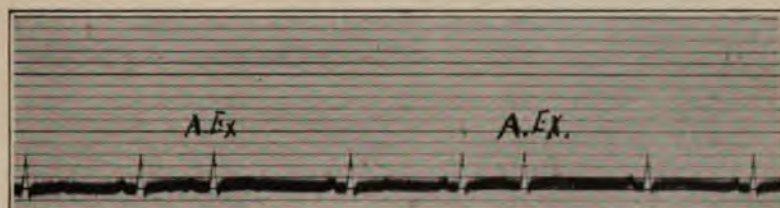


FIG. 95. — Auricular extrasystoles.

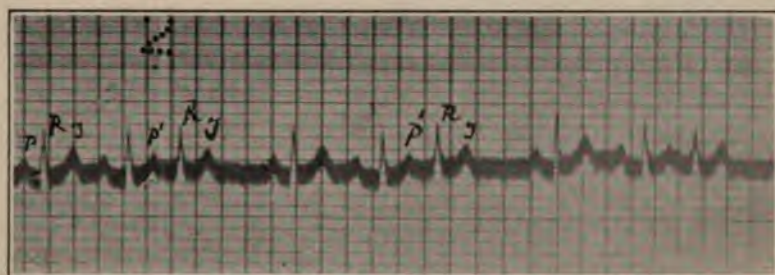


FIG. 96. — Auricular extrasystoles. The conduction time ($P'R'$) is slightly prolonged.

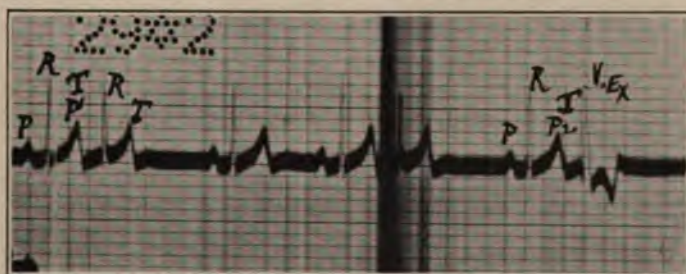


FIG. 97. — Auricular extrasystoles superimposed upon the T wave (TP'). The premature auricular contraction P' is followed by an ectopic ventricular beat ($V.Ex$).

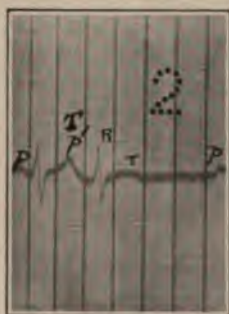


FIG. 98. — Auricular extrasystole showing the superposition of P and T waves ($P'T$).

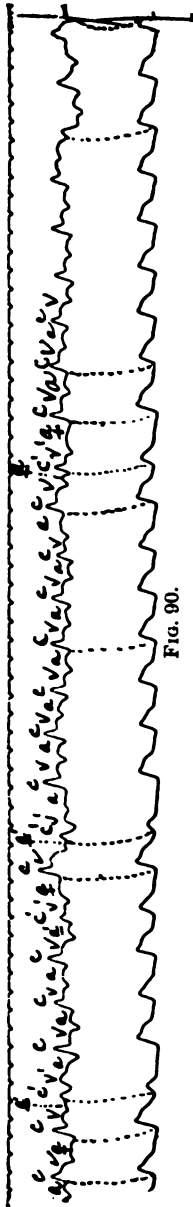


FIG. 90.



FIG. 91.

FIGS. 90, 91. — Auricular extrasystoles with shortened conduction time ($\sigma'\sigma'$). There is lengthened conduction time ($\sigma-\sigma$ in interval, Fig. 62) in some of the rhythmic beats.



FIG. 92. — Auricular extrasystole ($\sigma'\sigma'$). Lengthened conduction (σ) of the rhythmic beat.

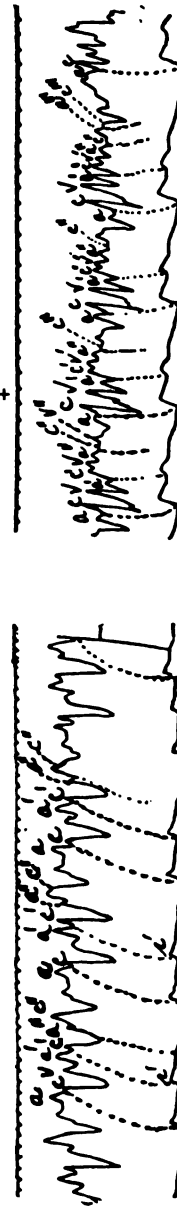


FIG. 93.

FIG. 94.

FIGS. 93, 94. — Multiple auricular extrasystoles. The radial tracing resembles coupled rhythm, but the frustrane contractions are well seen in the jugular tracing ($\sigma''\sigma''$, $\sigma'''\sigma'''$).

easily diagnosed; when interpolated, their recognition is more difficult. If the premature contraction is frustrane, that is, if its course in the ventricle is improperly directed or not sufficiently powerful to open the aortic valves, there is no corresponding pulse beat at the wrist; otherwise, the extrasystole causes a stronger or weaker pulse. At the heart one or two sounds of varying intensity accompany the premature contraction. If the extrasystole opens, and the aortic valves subsequently close, a premature first and a premature second sound is heard; if the ventricular pressure remains lower than the aortic, a premature first sound alone is heard. Only rarely is this sound too faint to be audible. While graphic methods of registration are required to determine the exact length of the compensatory pause, distinct shortening of the latter is readily determined by palpation at the wrist. Marked differences in the duration of the compensatory pauses characterize auricular, rather than ventricular, extrasystoles. The likelihood of a premature contraction being auricular is increased if it produces a fairly strong pulse beat; the weaker wave is more characteristic of a ventricular premature contraction. It is occasionally possible to distinguish auricular from ventricular extrasystoles by observation of the jugular pulsations, for in the ventricular type a large summation wave (corresponding to the polygraphic *a* and *c'* waves) may occasionally be seen.

Coupled Rhythm. — Its diagnosis rests upon the clinical recognition of regularly recurring extrasystoles. The distinction from coupled rhythm accompanying auricular fibrillation depends upon the clinical characteristics of the latter (*q.v.*).

Irregularly occurring extrasystoles of varying force in a heart beating rapidly are most apt to be confused with auricular fibrillation. The only differential guide is observation of the jugular and of carotid beats; even here, differentiation is often impossible because of the rapidity of the pulsatile waves which makes it extremely difficult for the eye to decipher them.

A II. PAROXYSMAL TACHYCARDIA OF AURICULAR ORIGIN

The term Simple Tachycardia should be applied to the common acceleration of the normal sinus rhythm; the usual ventricular rate is

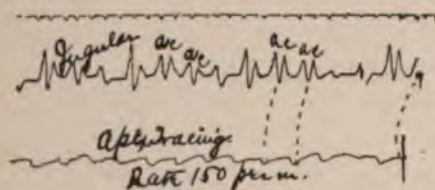


FIG. 100.

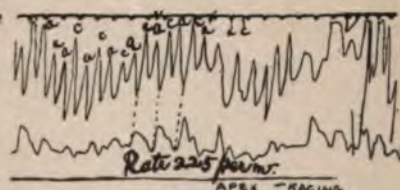


FIG. 101.

FIGS. 100, 101. — Varying ventricular rates from a case of exophthalmic goiter recorded on different days. When the rate is 150 per minute (Fig. 100), the *a-c* interval is normal. When the rate is 225 (Fig. 101), the *a-c* interval is slightly diminished; the diastole is entirely abolished. The *a* wave falls with the *s* of the preceding beat.

between 110 and 130 per minute. **Paroxysmal tachycardia** of auricular origin consists essentially in auricular extrasystoles coming in **attacks**. It is accompanied by an extreme, abrupt acceleration of the normal rhythm, with an **abrupt termination** and return to the normal. It may last minutes, hours, or, more rarely, days. Like the single auricular extrasystole, it is basically due to abnormal passage of the impulse. Graphically studied, the attack is seen to begin with a definite "**onset**" and to end with a definite "**offset**." Typical attacks are initiated and terminated by extrasystoles.

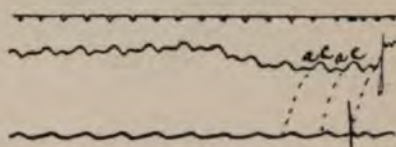


FIG. 102. — Paroxysmal tachycardia. Onset and offset not recorded. Shortened conduction time. The a wave falls with the preceding s.

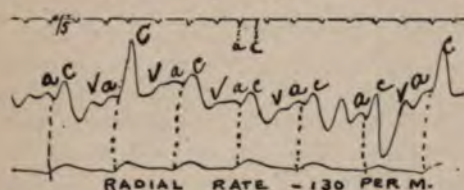


FIG. 103. — Exophthalmic goiter. Ventricular rate, 130 per minute. Very much shortened conduction time (a-c interval) despite only moderate acceleration (simple tachycardia).

ophthalmic goiter, I found diminished auriculo-ventricular conduction time despite the fact that ventricular acceleration was not extreme (Figs. 103, 106).

Studied electrocardiographically, it is found that the paroxysms usually originate from an **ectopic** auricular focus. When the change in the auricular complex is not marked, an origin close to the normal sinus area is assumed. When deviated negatively, it indicates an origin in the lower part of the auricular musculature.

Clinical Recognition of Simple

Tachycardia and of Paroxysmal Tachycardia of Auricular Origin. — The former refers to rhythmic pulse and ventricular action at the rate of about 120 or more per minute. Its recognition is usually simple.



FIG. 104.

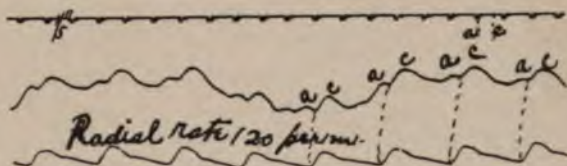


FIG. 105.

FIGS. 104, 105. — Exophthalmic goiter. Ventricular rate, 120 per minute; simple tachycardia; shortened a-c interval.

It may be confused with the rapid heart action of auricular fibrillation, but the pulse of the latter will be found arrhythmic upon longer or shorter observation. **Paroxysmal (auricular) tachycardia** is recognized

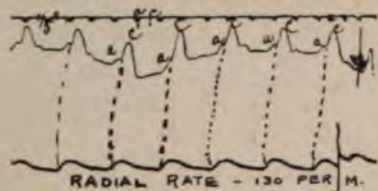


FIG. 106. — Exophthalmic goiter. Ventricular rate, 130 per minute; simple tachycardia; *a-c* considerably decreased.

by its characteristic **onset** and **offset**, an extrasystole with its compensatory pause initiating and completing the typical attack. These data, however, can rarely be obtained by direct clinical observation. In most instances we shall have to depend upon the history of a sudden beginning and termination of the attack; in addition, we may obtain a fairly accurate description of the occurrence of an extrasystole by such accounts as: "The heart stops for a moment," or: "There is a momentary faint feeling in the chest before or after the palpitation." Immediately or very soon after the attack, moderate tachycardia or even extrasystoles can often be evoked by such maneuvers as rapid breathing, exercises, sudden change of position, etc.

The differentiation of paroxysmal tachycardia from auricular flutter (*q.v.*) and from the tachycardial attacks accompanying auricular fibrillation (*q.v.*) will be discussed under those arrhythmias.

A III. AURICULAR INCOÖRDINATION

It will be noted that the three types of arrhythmia to be described — fibrillation, flutter, and the intermediate form — result primarily from auricular incoördination. An extreme degree of inchoate auricular activity results in auricular fibrillation. When incoördinate activity is less marked, it results in auricular flutter (or auricular tachysystole). There exists, also, an intermediate form.

A III. (1) AURICULAR FIBRILLATION

This constitutes approximately three quarters of all kinds of arrhythmias. We now know that the great majority of cases which Mackenzie called nodal rhythm belong to this category. When auricular fibrillation is typical, **ventricular activity** is **completely irregular** in rhythm and force, so that no two successive beats are alike. If induced experimentally in animals, the auricles show irregular tremulous fibrillating activity; sometimes and in some parts of the auricle the fibrillation is fine, and at other times and other places it is coarse. There is no unity in auricular contractility, although occasionally waves of fibrillation proceed with a fair degree of regularity over the entire musculature. The auriculo-ventricular conduction system is bombarded, as it were, by numerous irregular impulses, only some of which can pass through the

FIGS. 107-120. — Different types of polygrams of auricular fibrillation. The radial tracings show varying degrees of irregularity of rhythm and force of the pulse beats; some are grossly irregular (for example, Figs. 108, 114, 117); others more nearly approach the normal pulse rhythm (Figs. 113, 118, 120). The jugular tracings are of the most varied types. Their pathognomic characteristic is the absence of the rhythmic *a* wave regularly preceding the *c*. Small waves preceding the *c* are sometimes observed (Figs. 107, 108, 110), but their incidence and size are irregular. The *c* wave may be split (Figs. 115, 119) or may form a combined wave with the *v* (Figs. 118-120), especially when ventricular action is slow.

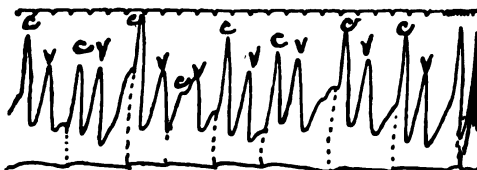


FIG. 107.

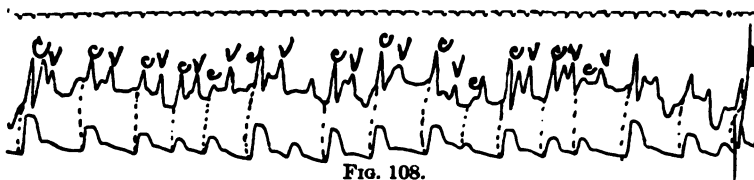


FIG. 108.

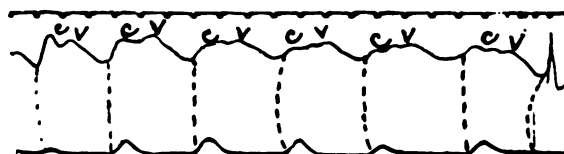


FIG. 109.

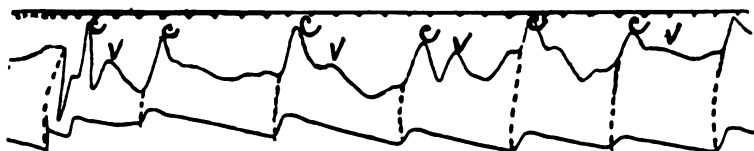


FIG. 110.

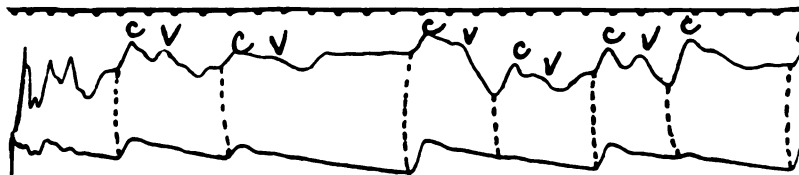


FIG. 111.

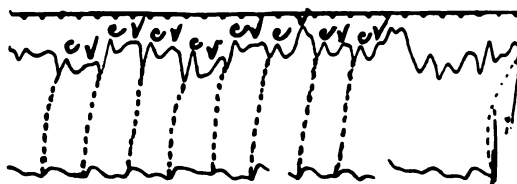


FIG. 112.

junctional tissue and excite the ventricle to irregular and discordant response.

The **polygram** corresponds to the pathological physiology just described. In typical instances the radial tracing shows gross and complete irregularity in the force and rhythm of the radial beats; in

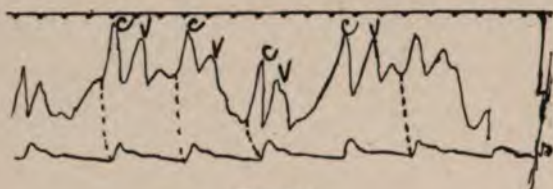


FIG. 113.

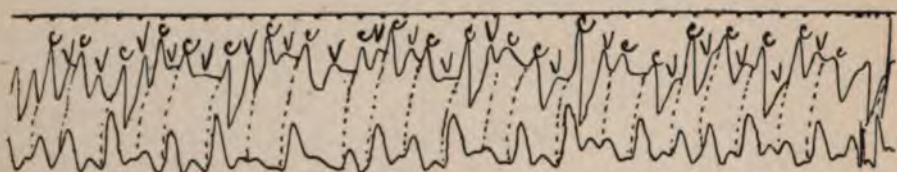


FIG. 114.

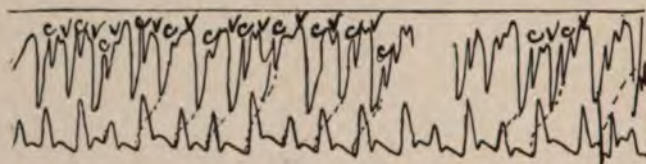


FIG. 115.

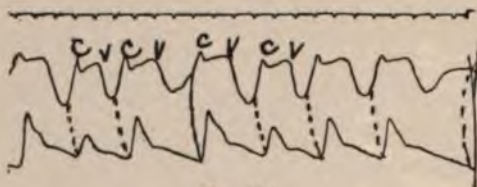


FIG. 116.

the jugular, the representative of orderly rhythmic auricular contraction, the *a* wave, is absent (Figs. 107-121). It is said that fibrillation is sometimes sufficiently coarse to produce small fibrillary reflux waves in the superior vena cava and jugular bulb; these are then indicated in the tracings as irregular wave-like lines. When found in mitral stenosis with a marked diastolic thrill, it seems to me that such fibrillary waves may be due, not to auricular fibrillation, but to turbulent diastolic ventricular eddies propagated as small waves from vibrations of the stiffened mitral valves, through the heart to the superior vena cava and jugular

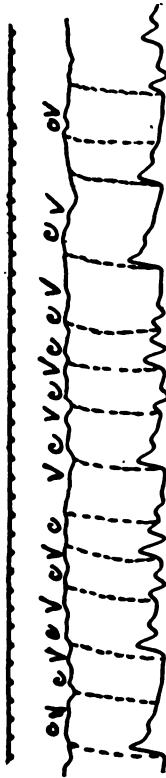


FIG. 117.

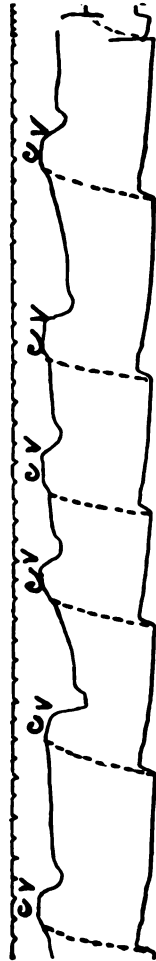


FIG. 118.



FIG. 119.



FIG. 120.

veins. Figure 122 shows fibrillary waves from a case of mitral stenosis with a loud, exceedingly rough, diastolic murmur and thrill. There was, in addition, a short, sharp, regularly recurring presystolic wave (Fig.

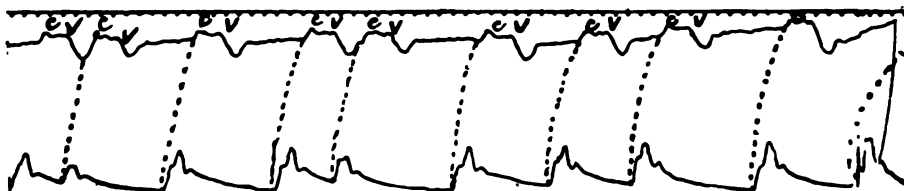


FIG. 121. — Auricular fibrillation, anacrotic radial pulse.

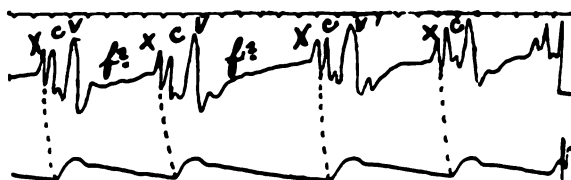


FIG. 122. — Auricular fibrillation with mitral stenosis showing presystolic (*x*) and fibrillary (*f*) waves.

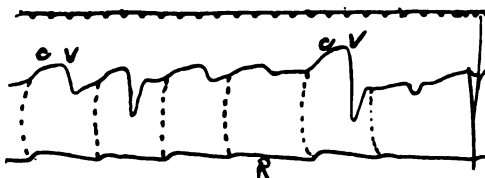


FIG. 123.

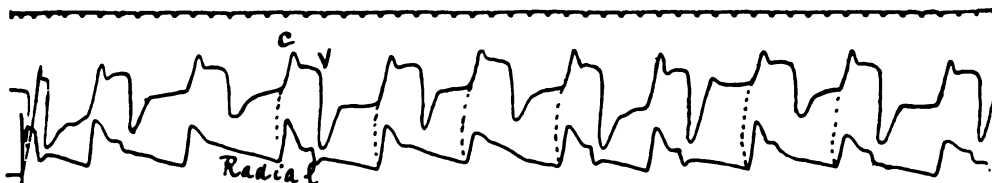


FIG. 124.

FIGS. 123, 124. — Auricular fibrillation; sharp fall of pressure following the ventricular filling wave. In Fig. 124, the radial pulse is quite rhythmical. Digitalis had been given.

122, *x*) whose etiology is not clear. Electrocardiograms taken at that time preclude the possibility of its being an auricular wave.

In some of the jugular tracings of auricular fibrillation, a sharp rise and subsequent fall of pressure succeeding the *r* wave is found (Figs. 123–124). As already indicated, a completely irregular pulse accompanies auricular fibrillation in the great majority of cases (Figs. 125–129).



FIG. 125.



FIG. 126.



FIG. 127.

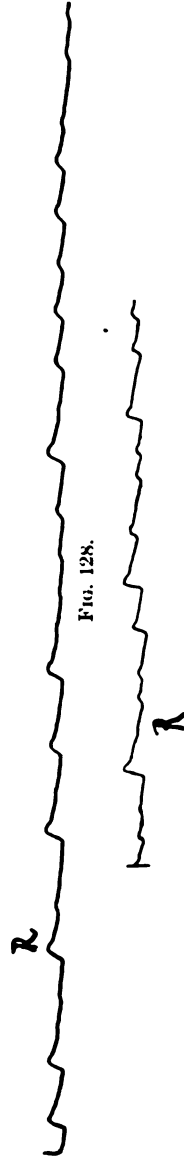


FIG. 128.



FIG. 129.

FIGS. 125-129. — Auricular fibrillation illustrating the usual types of accompanying pulse irregularities.

Very rarely the pulse becomes absolutely regular as the result of digitalis medication, though fibrillation continues (Fig. 124).

Corresponding to the state of incoördinate auricular activity characteristic of auricular fibrillation, there is in the **electrocardiogram** an **absence** of regularly recurring **auricular deviations** (*P* waves). In

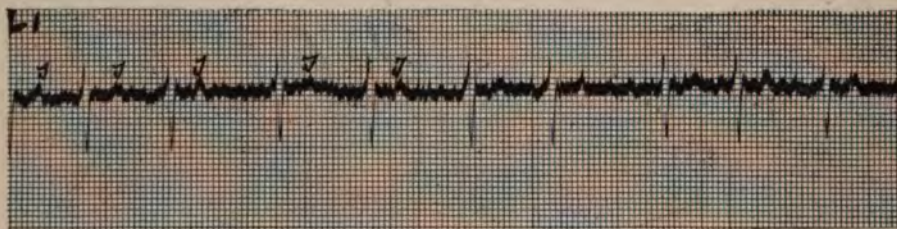


FIG. 133. — Fine fibrillation waves.

their place, there are undulations, representing fibrillation, at rates between 350 and 900 per minute. These fibrillation waves vary considerably in type. In short runs they may resemble the regular and rapid auricular activity of flutter; at other times the waves are coarse and arrhythmic, or are so small as to be scarcely distinguishable as separate deviations. Examples of these various types are shown in Figs. 130, 131 (Plate X), 132, 134, 135, 137 (Plate XI), 133, 136, 138, 139. At

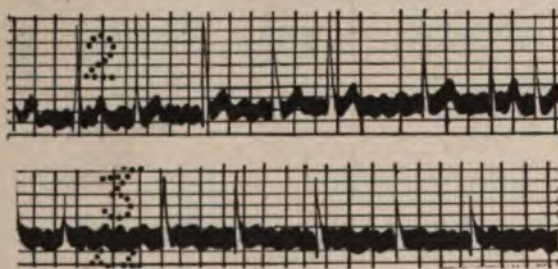


FIG. 136. — Fairly regular fibrillation waves in parts of *LII* and *LIII*.

present there is no clinical distinction based upon these differences.

In older people with cardiosclerosis, or as a result of digitalis medication in others, the pulse and heart action may be fairly regular in force and rhythm, so that graphic tracings are

required in order to establish the diagnosis of fibrillation. In another clinical type of fibrillation, the pulse rate varies between 180 and 225 per minute, and is small in volume and easily compressible. Upon palpation, the individual beats appear equal in force. Clinically, this resembles paroxysmal tachycardia. Electrocardiographically, it is distinguished from the latter by the absence of *P* waves and by the absence of an "onset" and "offset" (Fig. 137, Plate XI).

Ectopic beats are sometimes present in auricular fibrillation (Fig. 138, *Ec*). They are erroneously called extrasystoles; the latter term is properly applicable only to premature beats which disturb an otherwise

PLATE X

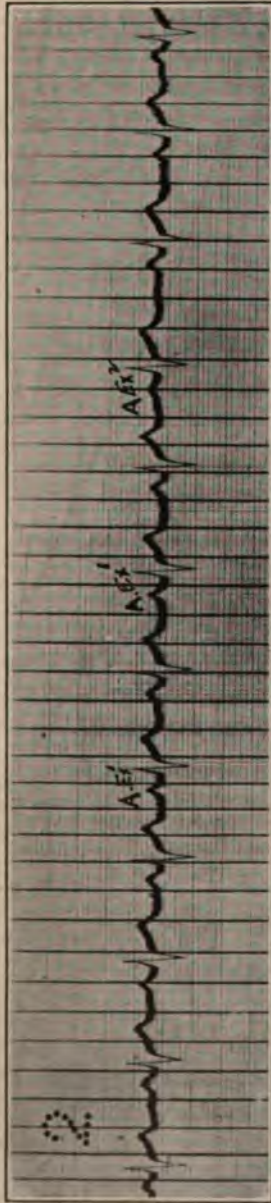


Fig. 99. — Auricular extrasystoles coming from two ectopic foci — *A.E.1*, *A.E.2*. These beats are neither premature nor followed by compensatory pauses.

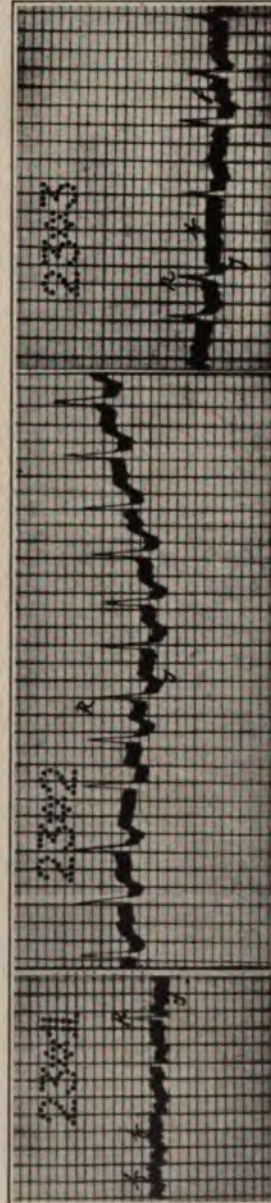


Fig. 130. — Small, fairly regular fibrillary (*f*) waves in parts of leads *I* and *III*. The extremely fine lines seen in *LI* and in some of the other electrocardiograms are due to an extraneous skin current, and not to fibrillation.

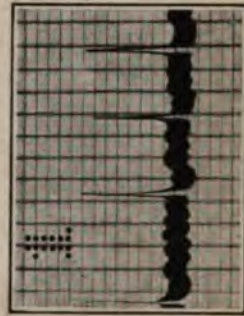


Fig. 131. — Fibrillation waves scarcely visible.

PLATE XI

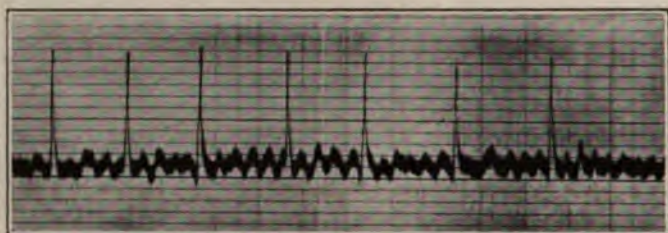


FIG. 132. — Fairly regular fibrillation waves which at times resemble flutter.

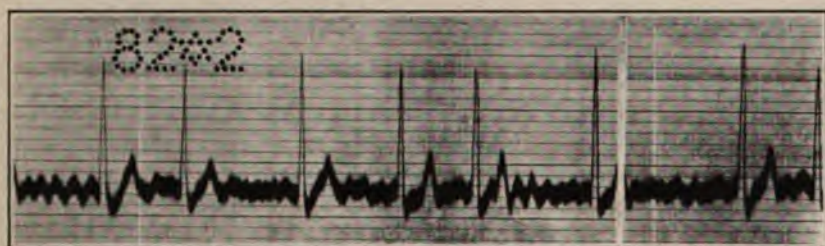


FIG. 134. — Various types of fibrillation waves, coarse and fine, in the same lead.

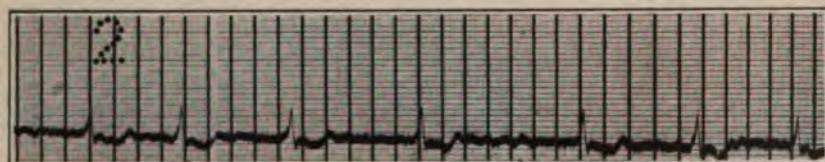


FIG. 135. — Very fine fibrillation waves.

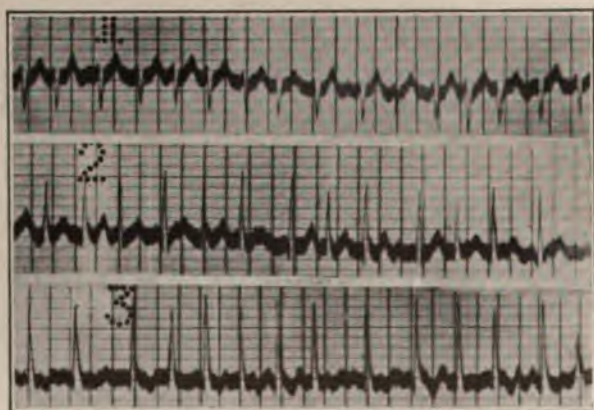


FIG. 137. — Auricular fibrillation showing tachycardia in *L I*. Note the varying rates of ventricular rapidity.

regular rhythm, and not to beats occurring in the complete irregularity of auricular fibrillation.

Transient Auricular Fibrillation. — Only exceptionally is it possible to note sudden transitions from normal rhythm to auricular fibrillation.

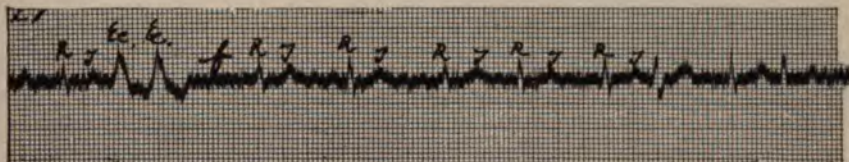


FIG. 138. — Auricular fibrillation with ectopic beat (*Ec*).

Such attacks may be the result of digitalis therapy. They are also found in exophthalmic goiter or at the crises of severe infections (for example, in pneumonia). They are by no means infrequent in older patients with cardiosclerosis, in whom hypertension is a marked clinical feature. Attacks of auricular fibrillation, in addition, occur in the decompensatory stages of valvular disease. Its occurrence with auricular flutter has already been pointed out. Its presence in a patient with no temperature and with no sign of organic cardiovascular disease is very rare. Figure 140

(Plate XII) is an example. It was taken from a child of eight, not neurotic, who had had several attacks of tonsillitis. Tonsillectomy was performed two years before. Shortly before she came under my observation, she suddenly felt her heart "jump": this "jumping" sensation has since been occasionally repeated. There were no gastric symptoms. The child was somewhat anemic and undersized. There

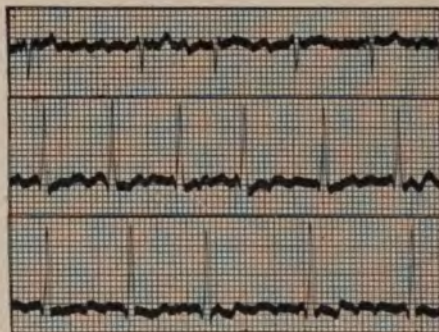


FIG. 139. — Auricular fibrillation with coarse fibrillation. (Courtesy of Dr. A. E. Cohn.)

were no signs of decompensation. The urine contained no abnormal elements. There was a very soft faint systolic murmur, of functional nature, at the apex; the murmur was not transmitted. The electrocardiogram (Fig. 140, *LIII*) shows the above-mentioned short run of auricular fibrillation (*A.F.* . . . *A.F.*) following the ventricular extrasystole (*V.Ex*). In addition, there is fairly marked sinus arrhythmia. There are several ventricular extrasystoles (*V.Ex*), in some of which the auricular beat is seen as a separate deviation (*P'*). There is also an auricular extrasystole of sinus origin with an abnormal ventricular complex (*LI, A.Ex*); it is not followed by a compensatory pause.

Clinical Recognition of Auricular Fibrillation. — The pulse and ventricular irregularity of **typical** auricular fibrillation is sufficiently obvious to be readily manifest. At the apex there is marked irregularity in force and rhythm of the heart's action. Sometimes scarcely two beats are alike; sometimes there is fairly regular heart action lasting several seconds or longer. At the wrist, if all beats come through, the pulse, completely irregular, takes on a helter-skelter characteristic. In the neck the carotids are correspondingly irregular; the jugular pulsations are usually too rapid to be individually identified. If there are many frustrane ventricular contractions (so-called pulse deficit), the radial seems fairly regular in force and rhythm, for it is mainly the small and weaker beats which produce the picture of complete irregularity; auscultation at the cardiac apex will readily reveal these small, numerous, and discordant beats. Indeed, in thin persons, **inspection** of the apical region shows the characteristically arrhythmic ventricular action of auricular fibrillation.

The **ectopic** beats in auricular fibrillation (sometimes erroneously called "extrasystoles") can occasionally be recognized even when ventricular action is rapid and irregular, because they are commonly followed by momentary (not compensatory) pauses, and by beats much louder and stronger than the preceding. In the **tachycardial attacks** occurring with auricular fibrillation, there is no onset or offset, the beats are sufficiently irregular in force and rhythm to be palpable and are apt to be interspersed with the typical gross irregularity of auricular fibrillation; after the attacks a completely irregular pulse is again present. These are data which serve in the differentiation between this type and the paroxysmal tachycardia already discussed.

In **auricular fibrillation with coupled rhythm** and fairly slow and regular ventricular activity and pulse, or in those rare instances of a perfectly regular pulse following digitalis medication, differentiation from the normal or from extrasystolic arrhythmia can only be made by direct observation of the jugular pulsations. If distinct auricular waves can be recognized, their presence serves to exclude fibrillation.

A III (2). AURICULAR FLUTTER. — AURICULAR TACHYSYSTOLE

In this type the auricles beat regularly and rapidly from 225 to 350 times per minute. Since the ventricle cannot respond at a like rate, heart block (*q.v.*), incomplete or complete, results. If the block be incomplete at a 2:1, 3:1, or 4:1 ratio, the pulse remains regular, the rate depending upon the ratio. If incomplete heart block be present with a constantly varying auriculo-ventricular ratio, the pulse becomes irregular. The **polygraphic recognition** of auricular flutter may be difficult, for the auricular waves are often small and distorted by respiration. Lewis has pointed out that under these circumstances the arrhythmic groups of radial beats form exact multiples, so that auricular flutter

PLATE XII

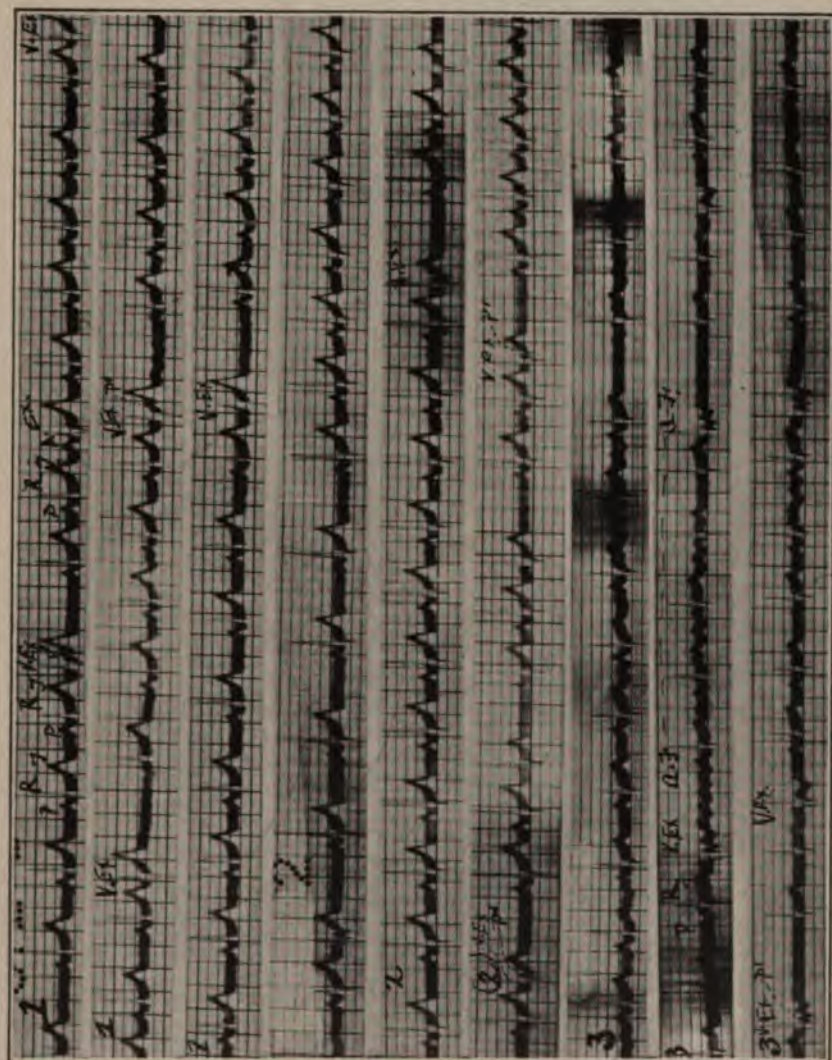


FIG. 140. — Various parts of a continuous electrocardiogram. The leads are indicated by the numbers. Several ventricular extrasystoles (Ex), some showing distinct auricular beats (P'), are seen. An auricular extrasystole of sinus origin and followed by an abnormal ventricular complex is shown in L I, A. Ex. In L III a short run of auricular fibrillation (AF . . . AF) is shown followed by a ventricular extrasystole (V. Ex).

may be diagnosed from the radial curve alone. I have not found this to apply to some cases of flutter that I measured and attempted to diagnose in this manner. When auricular waves are well marked (Figs. 141 *A* and *B*, Fig. 142), flutter is readily recognized. In the interpretation of the degree or type of block, it must be remembered that, when incomplete, the *a-c* interval may be abnormally prolonged; this may give the appearance of shortened conduction time, for the ventricle has

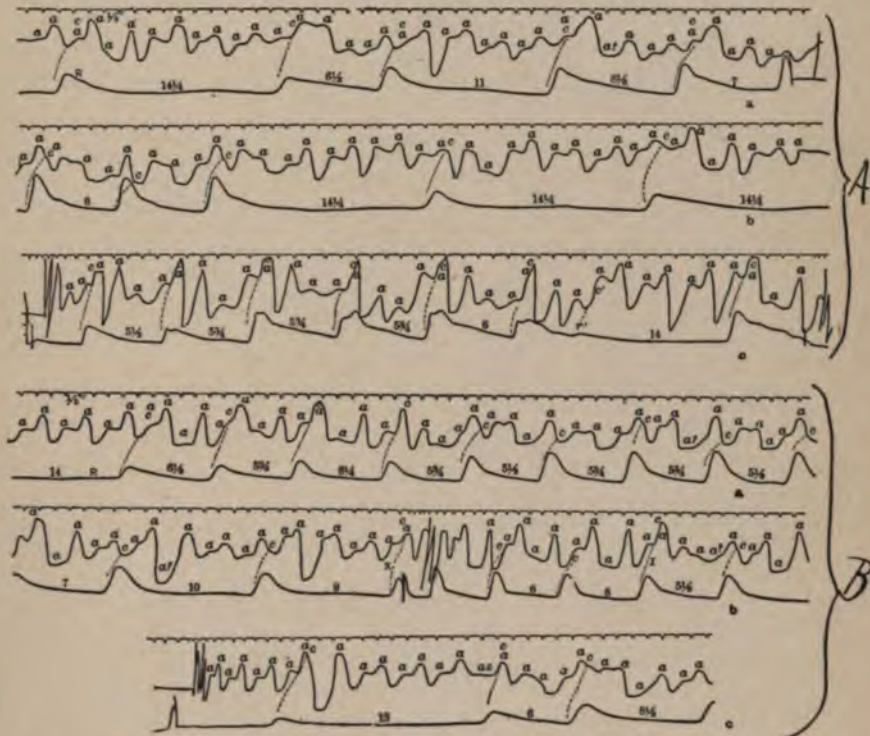


FIG. 141. — Tracing of auricular flutter. *A* and *B* were obtained on different days. *a*, *b*, *c* are continuous. In *A*, the auricular rate is 210; in *B*, 245 per minute. The numbers on the radial beats denote their respective lengths in $\frac{1}{4}$ seconds. The block is complete in *A* (*a* and *b*) and changes from incomplete to complete in *c*. The block is incomplete in *B* (*a* and part of *b*) and then later becomes complete.

probably responded, not to the immediately preceding auricular beat, but to the one previous (indicated by arrows, Figs. 142, 143). The conduction time is approximately .4 second. Partial heart block with a 3 : 1 rhythm is shown in Fig. 143. The pulse is almost regular throughout, though there are shorter and longer beats indicative of change in the auriculo-ventricular ratio. In other instances of flutter there may be found a change from partial to complete block, or the reverse (Fig. 141).

An **electrocardiographic** example of incomplete and complete block in the same electrocardiogram (Fig. 144, Plate XIII) is shown. Rapid, regular auricular activity is best seen in *LII* and *III*. The ventricular rate was 75, the auricular 300 per minute. Incomplete block at a 4 : 1 ratio was present. The *P* and *T* waves are occasionally superimposed. The *P* waves are contiguous and distinct in *LII* and *III*. They are scarcely discernible in *LI*. This is a characteristic of auricular flutter.

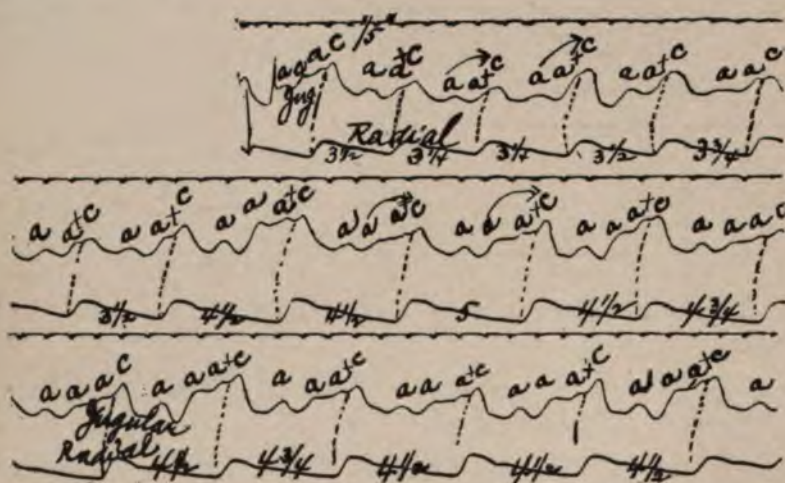


FIG. 142. — Auricular flutter. The numbers on the radial beats denote their respective lengths in $\frac{1}{2}$ seconds. The auricular impulse and its corresponding ventricular response are shown by arrows.

At a later date, the auricular speed was the same, 300 per minute. The ventricular rate varied from 60 to 100 per minute. There is no group proportion discoverable in the varying ventricular rates; complete heart block is present. After 10 drachms of the tincture of digitalis, given over a period of 10 days, there was no diminution of auricular speed; the ventricular activity was arrhythmic. There was also a difference in the heights of the *R* deviations. This difference was not of respiratory origin; it may have been due to differing degrees of ventricular excitability from digitalis medication. The electrocardiogram (Fig. 144, Plate XIII) was derived from a case of acute endo-pericarditis without decompensation; flutter developed during the course of acute rheumatic joint manifestations. In this instance, digitalis did not have the effect usually found in flutter with cardiac decompensation, viz.: first, auricular fibrillation; later, the resumption of normal rhythm with cessation of medication. As stated, there was no decompensation in this case. The rhythm became normal only when the joint symptoms subsided, the temperature reached the normal and the endocardial lesion became quiescent. From the clinical course, I conclude that the exciting cause

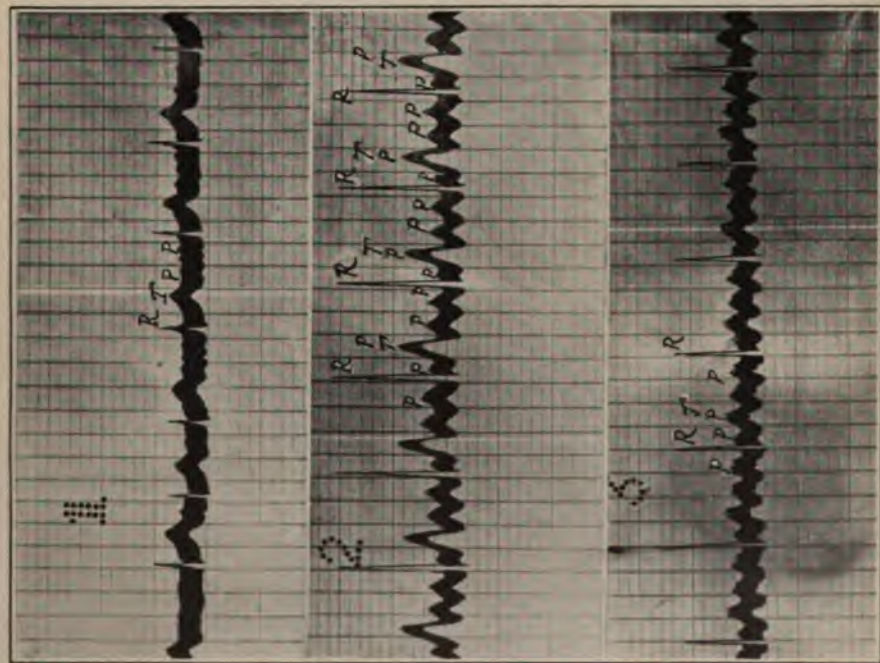


FIG. 144. — Auricular flutter, incomplete heart block, the ratio is 4 : 1. The auricular rate is 300 per minute; the ventricular 75. Note that the *P* waves in *LI* are almost indistinguishable; they are prominent and contiguous in *LII* and *LIII*.

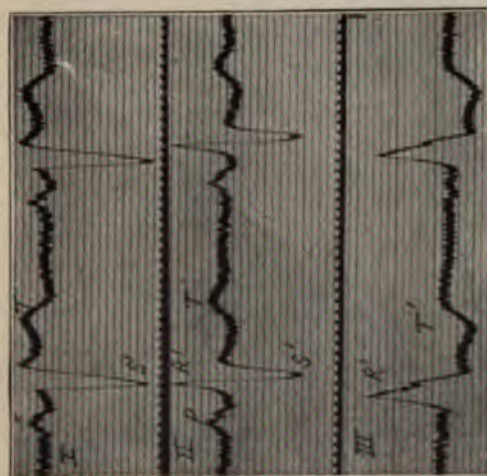


FIG. 103. — Lesion of the left branch of the conduction system. Conduction takes place along the right branch of the bundle. The initial deflections are tall and positive in *LIII*, and tall and negative in *LI*. (From E. P. Carter: Arch. Int. Med., 1914, XIII, 827.)



FIG. 177.—Electrocardiogram showing sinus arrhythmia of respiratory origin. Note the varying lengths of the diastolic pauses. (Courtesy of T. Stuart Hart: Abnormalities of Myocardial Function.)

of the flutter was the acute endocarditis. In what manner the latter produced the arrhythmia it is impossible to state. One year after the onset of the disease, the rhythm was still normal, the signs of the endocardial lesion (mitral regurgitation) were the same.

Clinical Recognition of Auricular Flutter. — In the exceptional instances of this arrhythmia in which the veins of the neck are tremendously distended by each auricular systole, the diagnosis becomes at once evident on inspection and estimation of the rapidity of the jugular pulsations. The latter, however, are more often minute and scarcely distinguishable as separate beats. Though not thus definable as individual waves, the jugular pulsations are often sufficiently distinct for one to note that their number greatly exceeds that of the ventricular contractions. In this manner the diagnosis of flutter may occasionally be ventured; but most diagnostic reliance must finally be placed upon recognition of heart block with the varying auriculo-ventricular ratios typical of flutter. If, for example, incomplete heart block at a constant ratio is present, the pulse and heart action are regular, the rate usually being between 90 and 120. If the auriculo-ventricular ratios be inconstant and constitute changing multiples, as from 2:1 to 4:1 or the reverse, the diagnosis of flutter can be made from the fact that the ventricular pauses also constitute multiples. If the auriculo-ventricular ratio constantly varies, or if complete heart block is present, ventricular action and pulse become arrhythmic, and clinically resemble the irregular action of frequent extrasystoles or of auricular fibrillation. The differentiation can then only occasionally be made from the latter, by the absence of its characteristic jugular pulse; from the former, by the absence of compensatory pauses.

A III. (3) INCOORDINATION INTERMEDIATE BETWEEN FLUTTER AND FIBRILLATION

In a few instances, I have observed a state of irregular auricular activity which may be regarded, I believe, as an intermediate stage of incoordination between flutter and fibrillation. The auricular rate was usually between 110 and 150; the beats, as shown by the differences in the auricular complexes, came from many scattered ectopic foci; the auricular rhythm was irregular. The ventricular rhythm was also irregular, the rate usually between 100 and 120. Many of the ventric-



Fig. 143 — Auricular flutter — partial heart block. The occasional difference in the lengths of the radial are due to changes in the ratio of auriculo-ventricular rhythm or possibly to differences in conduction time.

ular electrocardiographic complexes varied in the length of their deviations; very few ventricular extrasystoles were present; the picture betokened impulses following many vicarious paths in the ventricle. Although the electrocardiographic picture of the ventricular arrhythmia was not as distinct as was the case with the irregular (ectopic) auricular activity, it seemed probable that ventricular arrhythmia was likewise due to incoördinate activity, but of a degree considerably less than in ventricular fibrillation (*q.v.*).

The patients who presented this rare and interesting arrhythmia were in the agonal stages of pneumonia or of cardionephritis. It seemed possible, from the clinical pictures presented, that several factors

deserved etiological consideration: *e.g.* in pneumonia, the toxins; in cardionephritis, retained abnormal products. In all, there were probably changes in the intracardiac circulation which profoundly affected the nutrition of the general musculature, as well as of the auriculo-ventricular conduction system.

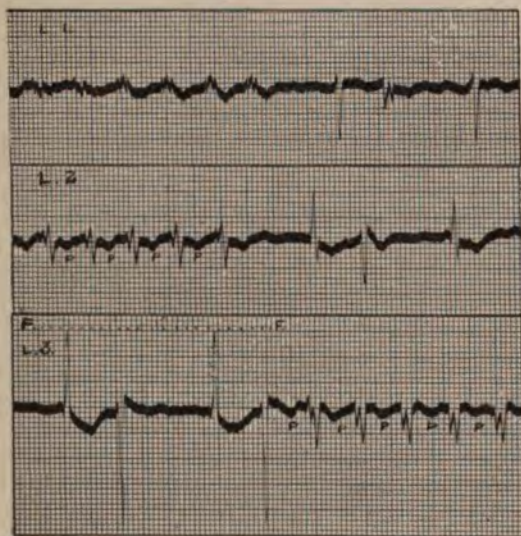


FIG. 145.—Paroxysmal auricular tachycardia. *F...F* (*L III*) shows change of paroxysmal tachycardia to auricular fibrillation (absence of regularly recurring auricular complexes). (Courtesy of Dr. A. E. Cohn.)

Although, as indicated, irregular auricular activity may be due to two fundamentally different causes (abnormal passage of impulses and auricular incoördination), it is interesting to note the gradations

and transitions in auricular irregularity. First are the isolated **auricular extrasystoles**. These, if numerous and originating outside the sinus area, give rise to **auricular tachycardia**. The rate is then between 150 and 225. If auricular speed be increased from 225 to 350 per minute, **flutter** results. This appears to be the limit of regular auricular activity in man. Any further increase in rate results in irregular activity—**auricular fibrillation**. The fibrillation waves may be as frequent as 900 per minute. In some instances it is possible to observe a transition from auricular flutter to fibrillation (Fig. 145). Between flutter and fibrillation is the **intermediate** type of inchoate auricular activity just described.

A'. NODAL EXTRASYSTOLES

Premature contractions having their origin in the auriculo-ventricular node are called nodal extrasystoles. The auricular and ventricular impulses start simultaneously, or almost simultaneously, from their nodal origin. Hence, in the jugular tracings, the *a* and *c* waves, and, in the electrocardiogram, the *P* and *R* waves fall coincidentally, or nearly coincidentally (Fig. 146). In the phlebogram, a large summation *a + c* wave is produced (Figs. 147, 148); in the electrocardiogram, the small *P* is often lost in the larger *R* complex.

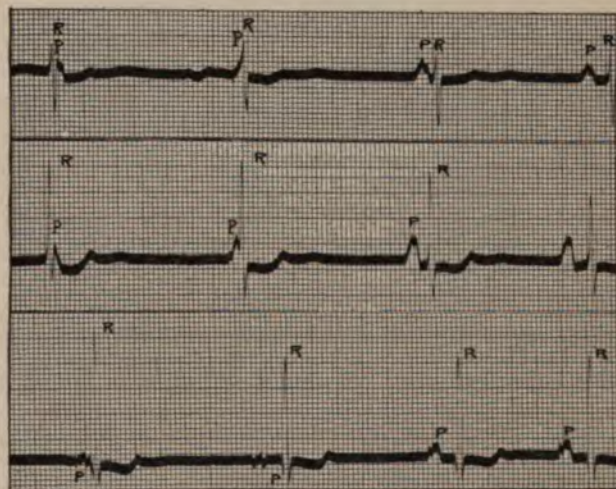


FIG. 146. — Nodal extrasystole showing coincidence of *P* and *R* waves. (Courtesy of Dr. A. E. Cohn.)

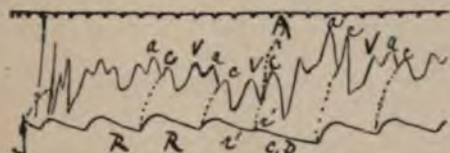


FIG. 147. — Nodal extrasystole. This is graphically illustrated by the almost synchronous occurrence of the foot points of *A'c'*.

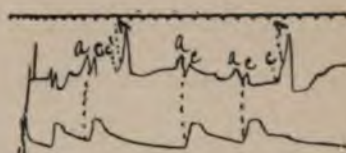


FIG. 148. — Nodal extrasystole which has produced no pulse wave. (Frustrane contraction.)

B. VENTRICULAR ARRHYTHMIAS

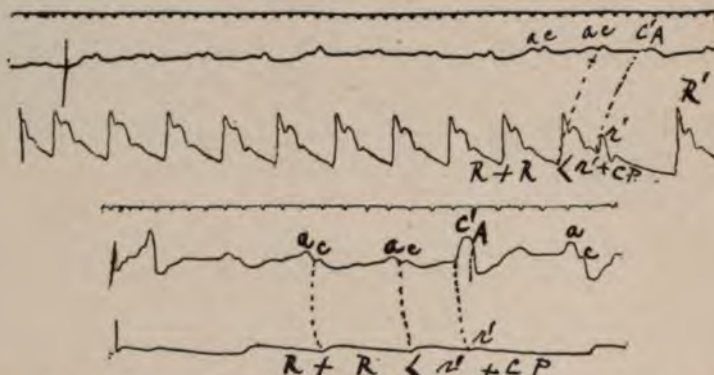
I. VENTRICULAR EXTRASYSTOLES

As with the auricular type, ventricular extrasystoles depend upon the **abnormal passage** of the impulse.

The main distinction between heterogenetic (aberrant) ventricular beats and those arising in the auriculo-ventricular node — true idio-ventricular rhythm — is one of rate. This node originates rhythmical contractions at a frequency between 25 and 40 per minute (heart block).

The rate of extrasystolic ventricular rhythm is between 130 and 200 per minute.

In **ventricular** extrasystoles (Fig. 82) the auricle follows its normal rhythm and contracts when the ventricle is in a refractory state, and,



FIGS. 149, 150. — The ventricular extrasystole plus the compensatory pause is slightly greater than two rhythmic beats. (Increased compensatory pause.)

as a result, there is no ventricular response until the next succeeding ventricular contraction. Thus the time of ventricular prematurity, added to the 'compensatory' pause, equals two rhythmic beats. This

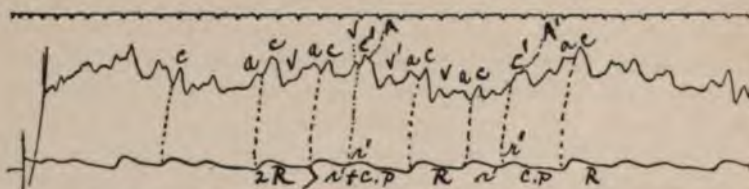


FIG. 151. — Ventricular extrasystole plus compensatory pause is less than two rhythmic beats (diminished compensatory pause).

fact is of value in the recognition of ventricular extrasystoles from radial tracings alone, or in polygraphic tracings in which the jugular is not sufficiently clear to be of value. In **auricular** extrasystoles, both cham-

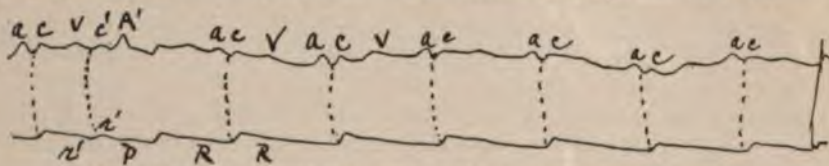


FIG. 152. — Ventricular extrasystole without compensatory pause.

bers contract prematurely and the succeeding pause may or may not be 'compensatory' (Fig. 81). Ventricular extrasystoles, likewise, are sometimes followed by pauses which are not exactly compensatory in

length (Figs. 149-152). Occasionally premature contractions occur so late that they lose their characteristic "prematurity" (Fig. 153). The amplitude of the radial beat following the extrasystolic pause in any

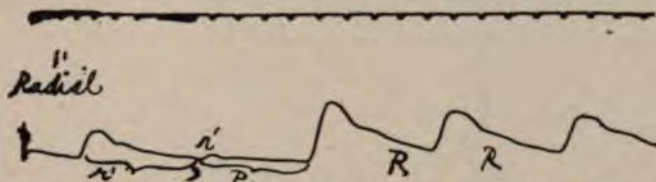


FIG. 153. — Extrasystole which is not 'premature.' r' is greater than P (the pause following extrasystole).

type of premature contraction is usually larger than that of the normal beat, and expresses graphically its greater strength (Figs. 83, 85, 153); this is sometimes regarded as indicative of favorable cardiac recuperative power.

B II. INTERPOLATED VENTRICULAR EXTRASYSTOLES

When the cardiac rate is slow, ventricular extrasystoles do not always interfere with the time of the next normal ventricular contraction; these are called "interpolated." This type of premature contraction occurs in the course of normal rhythm, and is not followed by a compensatory pause. Such extrasystoles are found in mid-diastole and in groups, rarely as isolated phenomena. Figure 56 (Plate VII) is therefore of interest because it shows a single interpolated extrasystole (*L II Ex*). The electrocardiogram was derived from a male patient 54 years old, who presented extreme decompensation. He had had syphilis 30 years before. The Wassermann blood reaction was positive. He had severe myocarditis and marked left ventricular hypertrophy. Fluoroscopy and radiography demonstrated aneurismal dilatation of the entire thoracic aorta. Under vigorous antiluetic, and the usual treatment for decompensation, the patient improved to such an extent that he now considers himself well. The electrocardiogram is of further interest because it gives some corroboration of the clinical findings: negative T in *L I* and an abnormally wide R deviation in *L I* and *III*, presumed evidences of myocarditis (Chapter IV); the positive $R I$ and negative $R II$ and $R III$ also confirm the clinical and orthodiascopic findings of a markedly hypertrophic left ventricle.

Electrocardiograms of ventricular extrasystoles vary considerably from normal complexes because their course in the heart (Fig. 38, *J, K*) is quite different from that followed by the normal excitation wave. The extrasystoles may originate in either ventricle. There is experimental evidence that extrasystoles arising from the various areas of the ventricular musculature — so-called ectopic foci (Lewis) — conform to definite electrocardiographic types. By analogy, it is possible to deter-

mine in the human being the abnormally excitable areas in the ventricle, from variations in the form of the atypical electrocardiograms (Fig. 154).

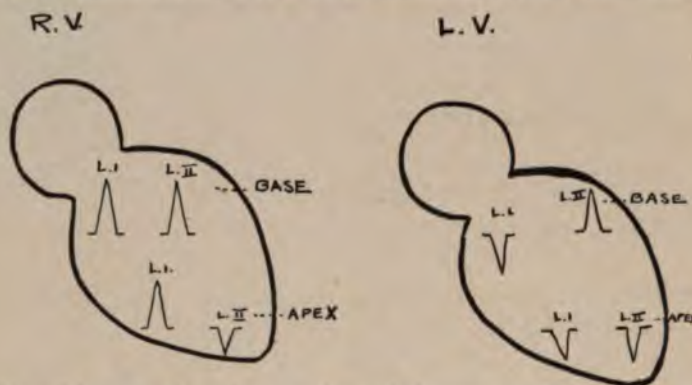


FIG. 154.—Schematic view of types of ventricular extrasystole arising from various ectopic foci, showing the direction of the corresponding *R* deviations.
R. V = right ventricle. L. V = left ventricle.

However, one must remember that the abnormal path followed by extrasystoles need not necessarily indicate a corresponding abnormal ventricular focus as its starting point.

Though studies of the various forms of ventricular extrasystoles are of considerable interest and importance, there is at present no correlation, except in isolated cases, between the different types and the clinical condition. It is to be noted that the *P* deviation is usually not visible because it is lost in the larger ventricular complex; occasionally, however, it appears at such time that it distinctly notches the

R wave.

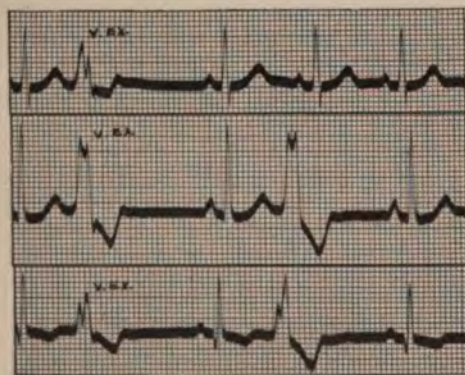


FIG. 155.—Ventricular extrasystole (*V. Ex.*) from right ventricle near the base. (Courtesy of Dr. A. E. Cohn.)

A schematic view of the usual ectopic foci and their corresponding electrocardiographic types is given in Fig. 154; it shows the direction of the *R* and *T* deviations in the *L I* and *II*. These types are also illustrated by examples of ventricular extrasystoles derived from human beings (Figs. 155, 156, 157). That there are intermediate types coming from intermediate ectopic foci one may

assume from certain rare cases of ventricular extrasystoles with heart block showing gradual and regularly varying abnormal complexes (Fig.

158). It is as if the abnormal excitation had affected, ladderwise and in progression, various successive areas of the ventricular musculature.

Instances of regularly recurring extrasystoles (coupled rhythm) are

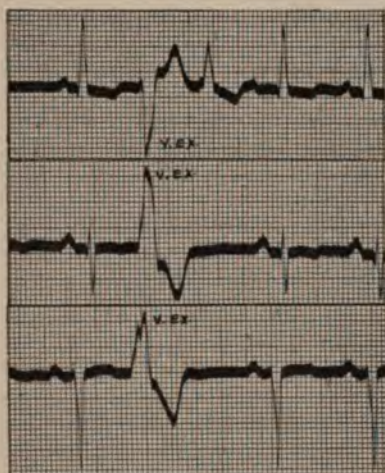


FIG. 156.—Ventricular extrasystole (V.Ex.) from ~~left~~ ^{right} ventricle near the base. (Courtesy of Dr. A. E. Cohn.)

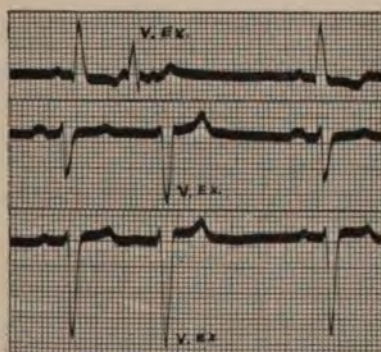


FIG. 157.—Ventricular extrasystole (V.Ex.) from wall of left ventricle. (Courtesy of Dr. A. E. Cohn.)



FIG. 158.—Extrasystoles arising from various ventricular foci, as shown by the differences in R complexes. Complete heart block present. (Courtesy of Dr. A. E. Cohn.)

given in Figs. 159, 159A, 159B. Occasionally several originate from the same ectopic focus (Fig. 160), producing short runs of paroxysmal tachycardia of ventricular origin.

B III. AUTOMATIC VENTRICULAR ACTIVITY — VENTRICULAR ESCAPE

This rare type consists in the sudden 'escape' of the ventricle from auricular control, and results in ventricular automatism or independent activity. In a few cases that had been previously described there was

marked retardation of the auricular, and, in most instances, of the ventricular rate. In the case which I observed there was never any marked pulse retardation (Fig. 161); the lowest rate was 55 per minute and the auricular speed was not abnormally slow. Its etiology will be discussed in a later chapter (Chapter VIII).

When auricles and ventricles beat at such rates that their waves and deflections in the tracings are regularly superimposed,



Fig. 159. — Regularly recurring ventricular extrasystoles (V. Ex.): — coupled rhythm (pulsus bigeminus). (Courtesy of Dr. A. E. Cohn.)

their origin in or near the auriculo-ventricular node has sometimes been assumed; these are called nodal extrasystoles (*q.v.*). Such simultaneous action is seen in parts of the electrocardiogram of the case of "ventricular escape" (Fig. 162, X, Plate XIV), but it apparently depends on transient identical auricular and ventricular speeds. For, as the latter vary, varying *a-c* and *c-a* (Fig. 161) or *P-R* and *R-P* intervals (Fig. 162, Plate XIV) soon occur. Besides, nodal beats are usually either regularly interpolated in the normal rhythm, or, when premature, are followed after longer or shorter compensatory pauses by the normal dominant beat.

Such assumptions of nodal extrasystoles are therefore not warranted, I believe, by the electrocardiograms here shown, all of whose complexes are alike. Backward conduction (*q.v.*) from ventricle to auricle, a rare reversal of the cardiac mechanism, also requires consideration as a possible explanation. In the case there described, the rhythm, when established showed a definite ventriculo-auricular conduction time similar to the normal, and was accompanied by marked ventricular slowing. This conception if applied to my case would

PLATE XIV

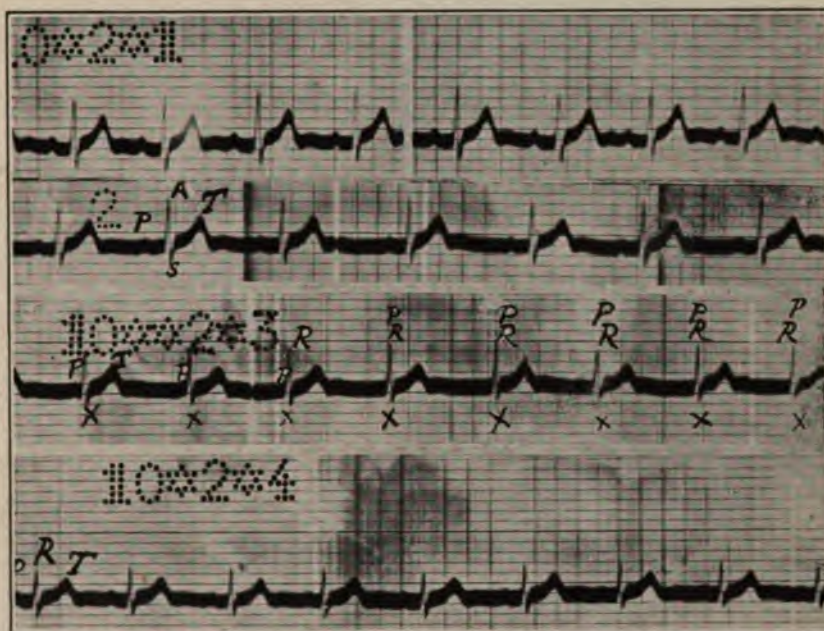


FIG. 162. — Ventricular escape. Sections of a continuous tracing. At X the ventricle "escapes" from auricular control and beats independently at such rate that auricle and ventricle occasionally contract synchronously (superposition of P and R waves). The rhythm is sequential in the other parts of the tracing.

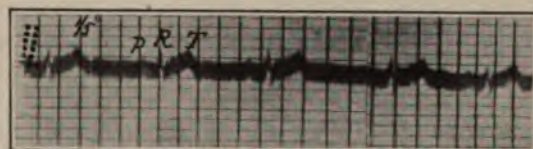


FIG. 169. — *LI*. True bradycardia. The auricles and ventricles beat sequentially. The rate is 50 per minute.



FIG. 183. — Prolonged conduction time. $P - R = .3$ second.

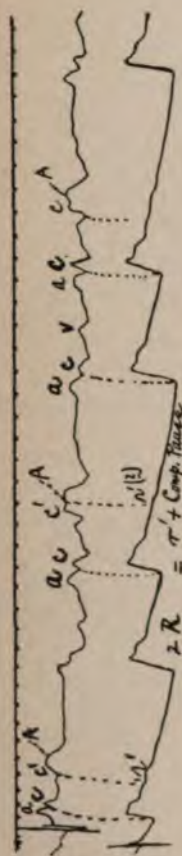


FIG. 159 A.



FIG. 159 B.

FIGS. 159 A-159 B. — Frustrate ventricular extrasystoles: coupled rhythm.



FIG. 160. — Short runs of paroxysmal tachycardia of ventricular origin (*Ex. 1, 2, 3, 4, 5, etc.*). A single ventricular extrasystole is seen at *Ex.* The auricles (*P* waves) beat regularly. (Courtesy of Dr. A. E. Collin.)

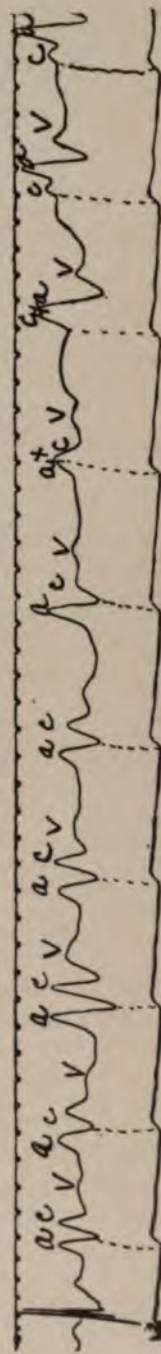


FIG. 161. — Ventricular escape. Part of a long series of tracings showing at times normal rhythm; at others, ventricular automatic activity (ventricular escape). Retrograde conduction from ventricle to auricle (backward conduction) could be excluded because of the irregular *c-a* intervals.

necessarily also assume that the "reversed mechanism" suddenly and irregularly ceased from time to time in those parts of the tracing which do not show a "retrograde conduction time" (*c-a* or *R-P* intervals) of less than one fifth second, and that, frequently, isolated beats were retrograde. This hypothesis is highly improbable and is not supported by the electrocardiogram. Rihl describes a case of occasional automatic ventricular action produced by vagal pressure. Lewis reports a case of rheumatic mitral stenosis with decompensation; digitalis had been given with resulting ventricular automaticity ("ventricular escape," Lewis). Gallavardin, Dufourt, and Petzetakis describe three cases with slow pulses (in one case the rate was 36 per minute) in which there was no clinical evidence of organic cardiovascular disease. In two, numerous polygraphic and electrocardiographic tracings showed the occurrence of ventricular automatism; in all three, it was readily evoked by ocular and vagus pressure, and by atropin injection. The writers suggest two causes for the phenomena: relative retardation of the auricular as compared with the idioventricular rate, or acceleration of the latter beyond the former. Two of their cases had very slow auricular rates occurring either spontaneously or induced by the methods described; the third showed no auricular retardation on vagus or ocular pressure, or after atropin injection. The arrhythmia in the first two cases was apparently due to relatively increased idioventricular rapidity beyond that of the sinus. Except for a very slight change in the complexes of the automatic ventricular beats in two of the cases — the absence of a very small *S* wave — all of the complexes are identical. In digitalis poisoning, Cohn and Fraser have occasionally found either identical auricular and ventricular speeds, or ventricles beating more rapidly than auricles, with ventricular escape. In the case I report there was at no time any marked pulse retardation — the lowest rate was 55 — nor was there any evidence of auricular slowing, although there was, at the periods of ventricular automatism, some slight difference between auricular and ventricular rapidity. Except for occasional somewhat slower beats, the idioventricular and normal ventricular rates were approximately the same. Slight sinus arrhythmia was sometimes present, but was not more marked than is frequently found as a physiological phenomenon.

B IV. PAROXYSMAL TACHYCARDIA OF VENTRICULAR ORIGIN

As with the auricular, paroxysmal tachycardia of ventricular type is marked by a rapid succession of ventricular extrasystoles (Fig. 160). This type of irregularity is rare. A paroxysm usually comprises from 8 to 10 premature contractions; very exceptionally, there may be as many as 25 or 30.

As distinguished from paroxysmal tachycardia of auricular origin, the runs of extrasystoles in the ventricular type are short; on auscultation, the strong "thumps" indicating extrasystoles are usually quite evident.

B V. VENTRICULAR INCOORDINATION

(1) **Ventricular Fibrillation**, similar to the incoordination of auricular fibrillation, is due to incoordinate ventricular activity. It has only very rarely been studied electrocardiographically in the human being. When it occurs, so far as we now know, it is followed by death within a very few minutes. The reason for this is that the inchoate ventricular contractions have no propulsive effect upon the blood in the ventricular chambers.

Branch-bundle Lesions.—The simplest type of ventricular incoordination consists of asynchronous ventricular contraction from lesions of the main branches of the auriculo-ventricular bundle. Such lesions may be permanent or transitory. In these lesions, the impulses originate in the *A-V* bundle before its bifurcation, and follow the unblocked branch in the ventricle (Fig. 38, *H, I*). Experimental and clinical examinations have shown that branch-bundle lesions produce characteristic electrocardiograms. Microscopic sections of post-mortem specimens have corroborated and confirmed the electrocardiographic evidence in a few instances.

Since the excitation wave travels along the healthy branch and excites the corresponding ventricle to contraction, the electrocardiogram in the various leads shows the characteristics of contraction of one ventricle alone, without the counterbalanced contraction of the

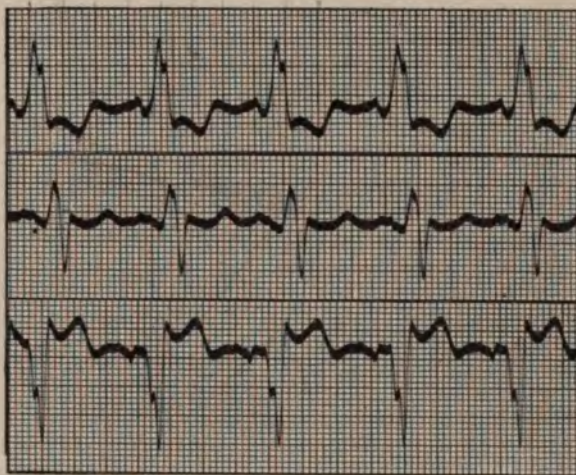


FIG. 164. — Lesion of the right branch of the conduction system. Conduction takes place along the left branch. The initial deflections are tall and positive in *LI*, and tall and negative in *LIII*. (Courtesy of Dr. A. E. Cohn.)

other. Hence, in lesions of the right branch, by far the most common, the initial deflections show left-sided conduction. The deviations are tall and positive in *LI*, and tall and negative in *LIII* (Fig. 164). The opposite is the case in the left-sided lesions (right-sided conduction) (Fig. 163, Plate XIII). A further characteristic of bundle lesions is the abnormal length of time required for the completion of the excitation wave, the width of the *R* usually being about .10 second. In addition, the deviations are frequently notched, the ventricular complex usually diphasic. The *T* wave is commonly deviated in a direction opposite to that of its accompanying *R* peak.

The electrocardiograms of cardiac hypertrophy are somewhat similar to those of branch lesions. They are differentiated by a narrower *R*, the lesser amplitude of their deflections, the direction of the *T* which is the same as that of the *R*, and by the tri- or quadriphasic character of the ventricular complexes.

C. TRUE BRADYCARDIA

This term should be applied only to types of slow, regular heart action in which there is **normal conduction time** from auricle to ventricle, and in which each best represents a **sequential auriculo-ventricular contraction**. Thus defined, it has its proper place in a classification of arrhythmias; otherwise, when loosely used in the sense of slow pulse rate (so-called spurious bradycardia), it takes no cognizance of auricular rhythm or of auriculo-ventricular conduction. It may thus haphazardly

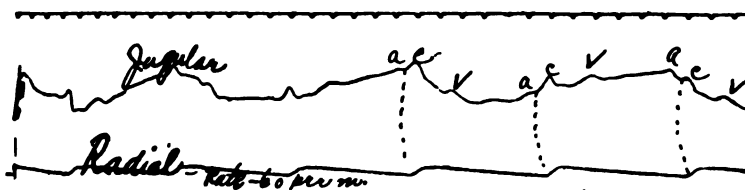


FIG. 165.

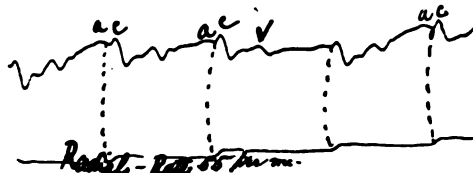


FIG. 166.



FIG. 167.

FIGS. 165-167. — True bradycardia from patients with gastric symptoms and normal hearts.

include such divergent cardiac irregularities as auricular fibrillation, extrasystoles, and heart block (*q.v.*). It is difficult to fix a definite rate as typical of true bradycardia. Cardiac and pulse rates ranging from 60 to 65 per minute are by no means rare in normal healthy adults, especially in those with rather marked vasomotor instability, flushed hands and face, sudden irregular pallor, and cold and moist extremities. Examples of regular, slow heart action due to extracardial causes are given in Figs. 167, 168, Fig. 169 (Plate XIV). An instance due to salicylate of soda is shown in Fig. 168.

In the electrocardiogram of true bradycardia, all the ventricular complexes are alike; there is slow, regular heart action. Thus, Fig. 169 (Plate XIV) taken from a young epileptic with a normal cardiovascular apparatus, shows regular sequential heart beats at the rate of 50 per minute.

Clinical Recognition of True Bradycardia.—The rate is rarely below 40 per minute; the cardiovascular apparatus is often organically normal. Since in true bradycardia the auricles and ventricles beat sequentially, the jugular waves (*a* waves) are seen to precede the carotid pulsations (*c* waves). True bradycardia must be differentiated from the

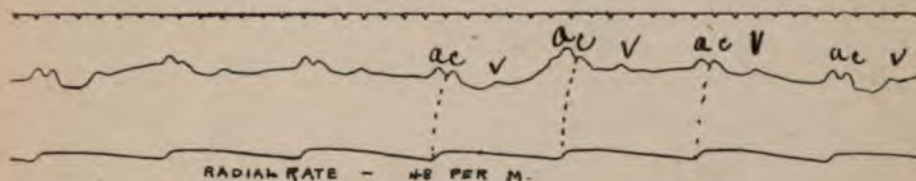


FIG. 168. — True bradycardia caused by salicylate of soda.

slow heart action found in heart block. In the former, the jugular pulsations occur with the same frequency as the carotid, the ventricular, and the pulse beats; in the latter, they occur more frequently. The jugular pulsations may be rendered more prominent by having the patient lie flat and by inspection of the right side of the neck. By holding a white card in the space between the jugular and carotid, it is sometimes possible to reflect the jugular pulsations on the card by placing the patient in the proper light.

D. ARRHYTHMIAS PRODUCED BY ABNORMAL SEQUENCE OF CONTRACTION OF AURICLES AND VENTRICLES

This sequence may be disturbed either at the **pacemaking area** (the sino-auricular node) or at the **junctional tissues** (the atrio-ventricular node).

D I. (1) SINUS ARRHYTHMIA

This pulse irregularity is produced both by physiological and abnormal influences which affect the sinus region, the pacemaker of the heart. Sinus arrhythmia of the **physiological respiratory** type consists of alternate moderate acceleration and retardation, a waxing and waning of the pulse rate, corresponding to inspiration and expiration respectively. The arrhythmia is of vagal origin and is ascribed to differences in the vagal inhibitory tone from phasic respiratory changes. It is a physiological phenomenon in children (Figs. 170, 171) and young adults (Fig. 172) (the "youthful irregularity" of Mackenzie), though it is by no means uncommon as a normal variation in the middle aged, especially upon forced deep respiration. Sinus arrhythmias due to other causes are also illustrated (Figs. 173-176).

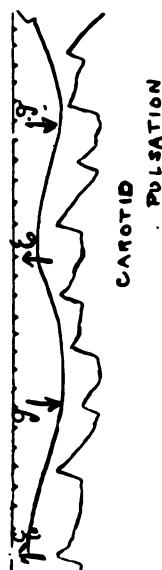


FIG. 170. — Carotid pulse of a normal child. The breathing curve with the inspiratory (*I*) and expiratory (*E*) phases, and corresponding difference in pulse rate are shown.

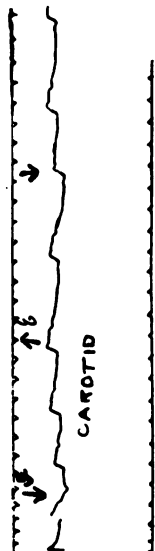


FIG. 171. — Moderate sinus arrhythmia (from a normal child).



FIG. 172. — Retardation (sinus slowing) from holding the breath at the end of inspiration. The arrow indicates the point at which normal breathing ceased (from a young healthy adult).



FIG. 173. — Slight sinus arrhythmia at the time of crisis in recovery from pneumonia.
I = inspiration; *E* = expiration.

PLATE XV

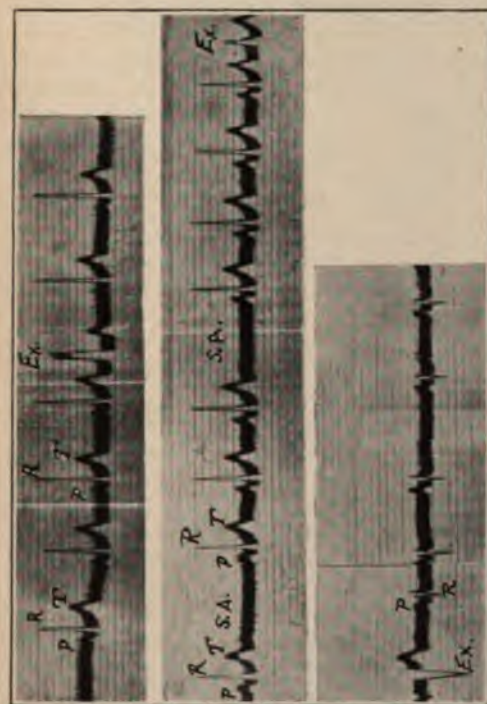


FIG. 179. — Electrocardiogram showing sino-auricular block (S.A. B) due to smoking.

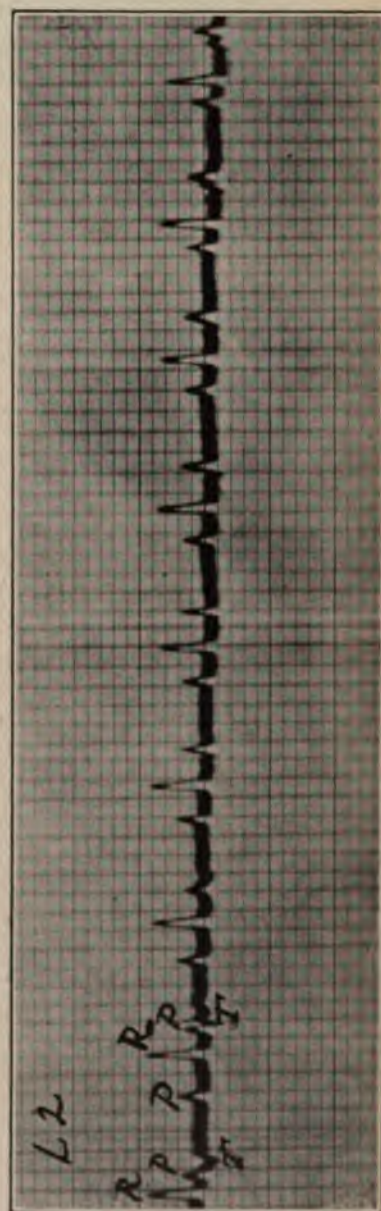
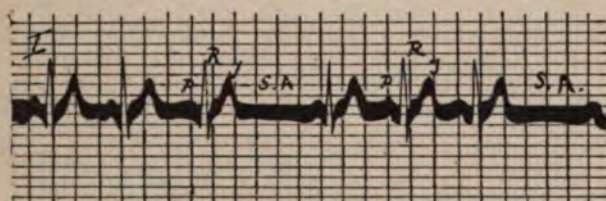


FIG. 197. — Complete heart block. The ventricular rate is 43 per minute; the auricular rate 75 per minute.

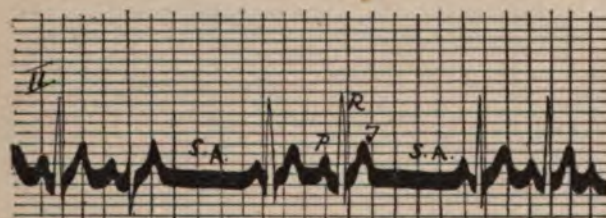
In the electrocardiogram, sinus arrhythmia is recognized by varying lengths of the pauses between the beats (Fig. 177); there is no difference in any of the complexes unless phasic respiratory variations (Chapter IV) are present.

Clinical Recognition of Sinus Arrhythmia. — The **physiological type** with normal pulse rate is readily diagnosed because it wanes and waxes with inspiration and expiration, respectively. The **pathological types** of sinus arrhythmia do not usually show this correlation. The clinical diagnosis of the latter is then made by the comparatively slow pulse rate with irregularly long diastolic pauses. The interventricular interval may be sufficiently long to block out an entire auriculo-ventricular contraction, thus leading to **sino-auricular block** (*q.v.*). The distinction between sinus arrhythmia of the non-respiratory type and auricular fibrillation with slow and fairly regular ventricular activity depends upon the observation of the jugular pulsations. In the former, there are regularly recurring jugular waves (*a* waves) which precede the carotid pulsations; in the latter this relationship naturally does not

exist, since auricular contractions are absent.



D I. (2) SINO-AURICULAR BLOCK



Occasionally abnormal influences affect the vagus or its endings in the sino-auricular node so that an entire beat is blocked.

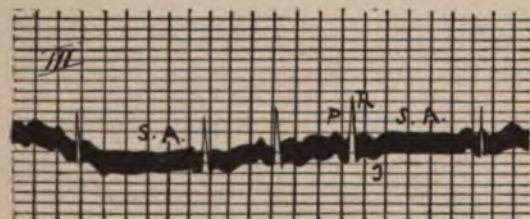


Fig. 178. — Electrocardiogram showing sino-auricular block (S.A.) due to smoking.

This arrhythmia is rare in adults. There is **stoppage of the entire heart** (Fig. 178: Fig. 179, Plate XV). The pause is usually somewhat less than is required

for two normal contractions; when extreme, the pause may represent the time required for three, or even four, normal beats. I have seen two cases due to tabagism, one of whom had an organically normal

PLATE XV

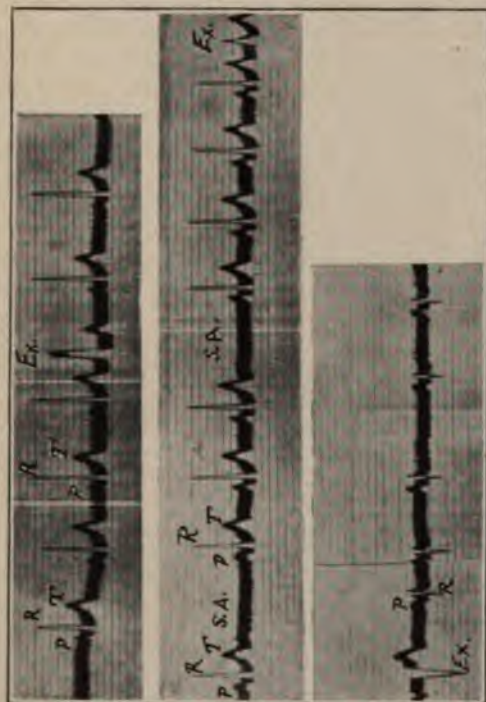


FIG. 179. — Electrocardiogram showing sino-auricular block (S-A. B) due to smoking.

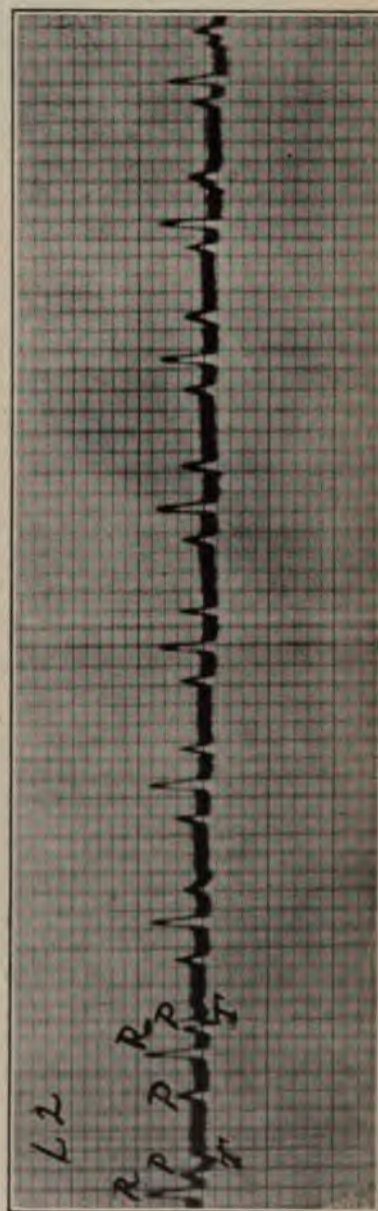


FIG. 197. — Complete heart block. The ventricular rate is 43 per minute ; the auricular rate 75 per minute.

heart; the other patient had myocarditis with mild decompensatory symptoms. Figure 178 is taken from one of these patients. In addition to the sino-auricular block, there is moderate tachycardia. This double effect of nicotine, moderate tachycardia and sino-auricular block, was probably due to the varying action of the poison upon the ganglionic terminations of the vagus and sympathetic (Fig. 180). In the other case, a smoker with myocarditis and decompensation, ventricular extrasystoles (Fig. 179, Plate XV) were also present; sino-auricular block disappeared two days after smoking was stopped; the extrasystoles ceased some days later when compensation was restored by the use of digitalis and theobromine sodium salicylate.

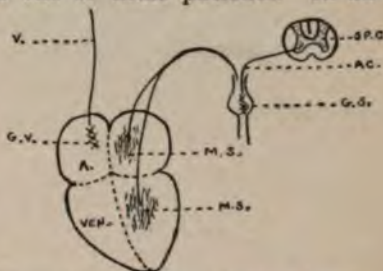


FIG. 180. — Diagram showing the effect of nicotine upon the ganglionic terminations of the vagus and accelerators. (Modified from Cushny's "Pharmacology.")

V = vagus;
A.C. = accelerator fibers;
Ven = ventricle;
A = auricle;
M.S. = cardiac musculature;
G.V. = ganglia at vagus termination;
G.S. = ganglia at termination of sympathetic nerve;
S.P.C. = spinal cord.

D I. (3) BLOCKED AURICULAR BEAT

Ventricular action does not follow the blocked auricular impulse, consequently there is a quiescent period equivalent to two beats (Fig. 181).

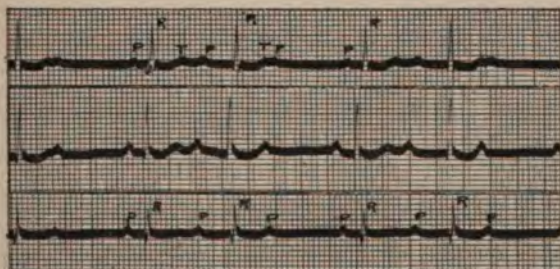


FIG. 181. — Blocked auricular beat. Absence of ventricular response, also showing prolonged conduction time. (Courtesy of Dr. A. E. Cohn.)

It is distinguished from extrasystoles by the absence of the characteristic small pulse wave and of the extrasystolic heart sounds; from sino-auricular block, only by recognition of an auricular jugular wave in the former, and its absence in the latter.

D II. (1) PROLONGED CONDUCTION TIME

This represents the **simplest type of heart block**. Various factors cause moderate prolongation of the impulse from auricle to ventricle, that is, of the P-R time. These are chiefly digitalis medication, myocarditis, and acute endocarditis. I have also observed lengthened conduction in several patients with auricular extrasystoles of functional

origin. Cases of extreme prolongation of the *P-R* time up to or even more than 0.50 second sometimes occur. For example, Fig. 182

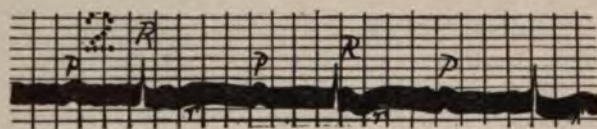


FIG. 182. — Electrocardiogram showing very slow ventricular rate (37 per minute) with a *P-R* time of .8 second.

is the electrocardiogram of a male patient aged 62, who had myocarditis and nephritis; at the time that the electrocardiogram was taken he

was suffering from a third attack of severe decompensation; dyspnoea, oedema of the legs, ascites. He died later with uremic symptoms. The electrocardiogram shows a ventricular rate of 37 per minute with a conduction time of 0.8 second. A less marked instance is shown in Fig. 183 (Plate XIV).

D II. (2) SHORTENED CONDUCTION TIME

The normal *P-R* interval is from .16 to .20 second. This interval — the conduction time from auricle to ventricle — is occasionally shortened in both paroxysmal and simple tachycardia; however, rapid cardiac activity in these arrhythmias occurs mainly at the expense of diastole.

D II. (3) BACKWARD CONDUCTION FROM VENTRICLE TO AURICLE

This exceedingly rare anomaly is exemplified in Fig. 184. It shows the auricular (*P*), regularly following the ventricular, beat. Backward conduction was also corroborated in that case by fluoroscopic examination. The patient, a laborer of 51 suffering from diarrhea for one

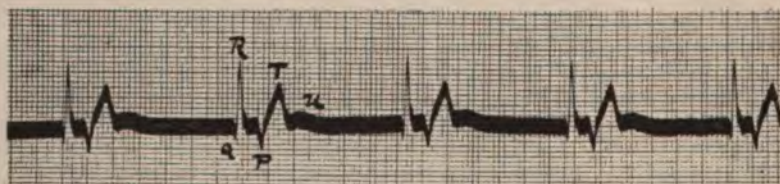


FIG. 184. — Electrocardiogram showing backward conduction from ventricle to auricle. (From Williams and James — "Reversal of the Cardiac Mechanism." *Heart*, 1913-14, V, 109).

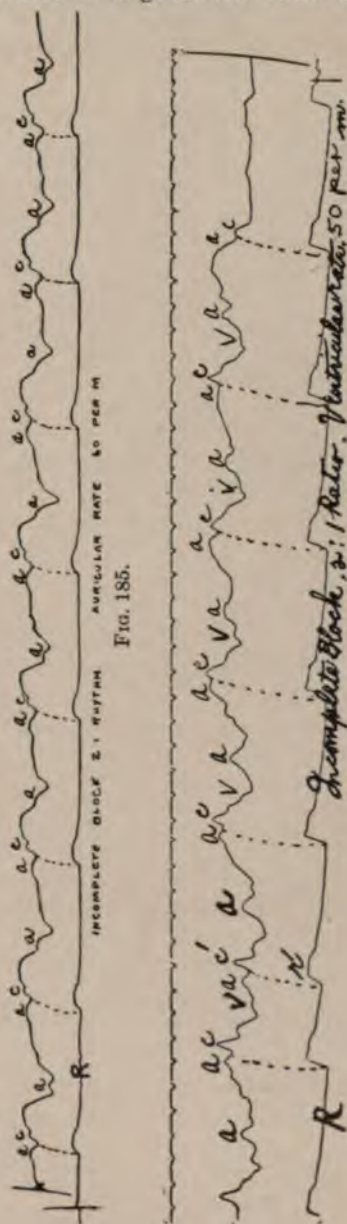
year, had attacks of Stokes-Adams syndrome. The cardiovascular system was apparently normal organically. The arrhythmia persisted for many months and was not affected by atropin injections. Finally, the rhythm again became normal. The writers suggest as possible etiological factors abnormal vagus control or a toxic cause.

D II. (4) (a) INCOMPLETE HEART BLOCK

In its simplest type, this consists in the absence of ventricular response to auricular impulses. There is a geometric ratio between auricular and ventricular beats; for example, the ventricle responds to every second, third, or fourth auricular impulse. Such cases are termed incomplete heart block at 2:1, 3:1, etc. ratio respectively (Figs. 185, 186). The ventricle and pulse beat rhythmically unless disturbed by occasional premature ventricular contractions (Fig. 186, *c'*). In other types of incomplete block the ventricular response varies irregularly from one ratio to another; the ventricle, for example, answers haphazardly every second or third or fourth auricular impulse. The pulse becomes correspondingly irregular. In both incomplete and complete block, the auricles beat rhythmically at approximately normal speeds, 60 to 80 times per minute. In incomplete heart block, the ventricular rate is considerably diminished; as stated, its regularity depends upon the auriculo-ventricular ratio.

D II. (4) (b) COMPLETE HEART BLOCK

This is said to exist when there is no relationship between auricular and ventricular speeds. The ventricle follows its own inherent rhythm (Figs. 187-196). The usual inherent (idioventricular) rate of the atrioventricular node is between 25 and 40 per minute. Complete heart block represents the purest type of



FIGS. 185, 186. — Incomplete block with a 2:1 ratio. In Fig. 186 there is a premature ventricular contraction (*c'*). The preceding auricular beat is not premature. The pause following *c'* is not compensatory, but is equal to an entire rhythmic beat.

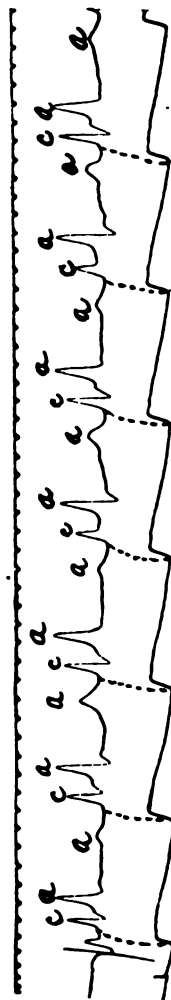


FIG. 187.

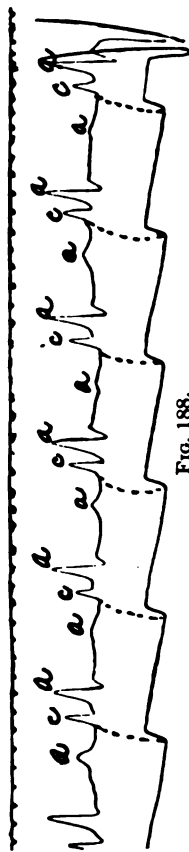


FIG. 188.

FIGS. 187, 188. — Complete heart block.



FIG. 189.

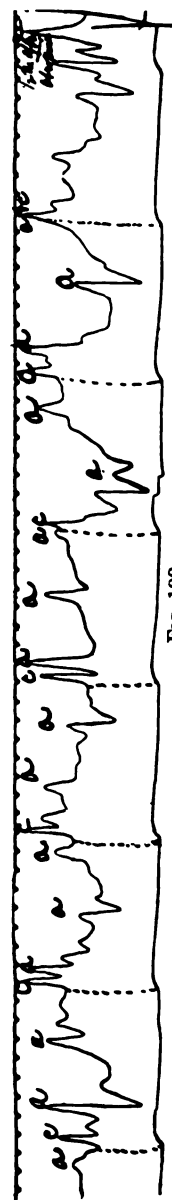


FIG. 190.

FIGS. 189, 190. — Complete heart block showing occasional superposition of a and c waves.

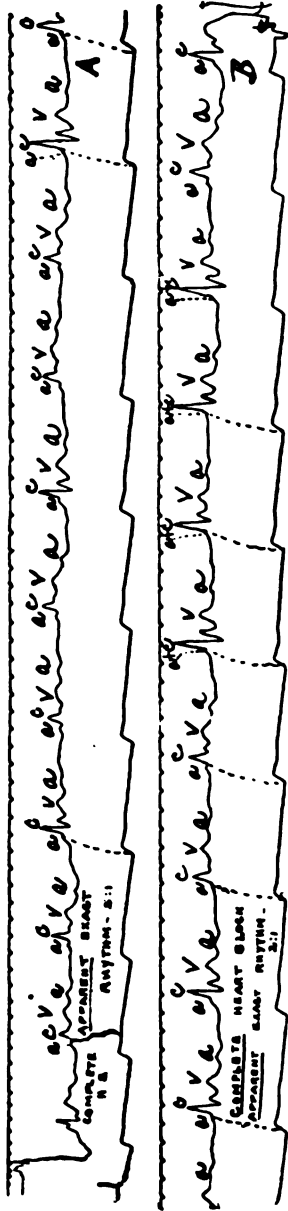


FIG. 191 A, B. — Complete block. In A the block is only apparently incomplete because the auricular speed is exactly twice that of the ventricle. In B, continuous with A, there is a slight variation in auricular and ventricular rates; the a-c interval becomes progressively smaller until a and c are superimposed, showing that the exact doubling of speeds and that the apparent normal a-c interval in A were purely accidental.

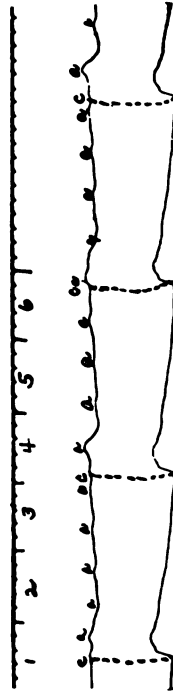


FIG. 192. — Complete block; ventricular rate 25 per minute; auricular rate 105 per minute. This tracing was corroborated by an electrocardiogram.



FIG. 193. — Complete heart block with irregular ventricular action. The shorter radial beats are underlined.



FIG. 194. — Complete heart block with a ventricular extrasystole. The ventricular rate is 27; the auricular rate is 81 per minute. The pause following the extrasystole is not compensatory; it equals a rhythmic beat.



FIG. 195 A.

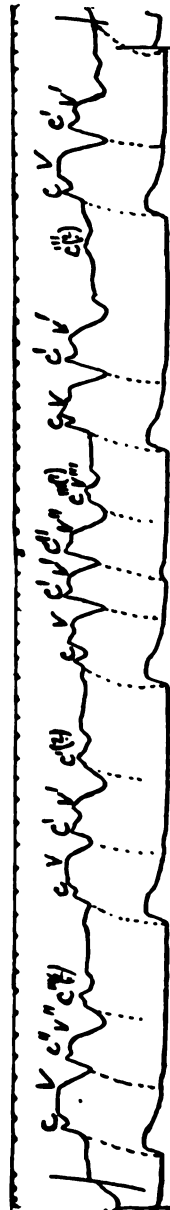


FIG. 195 B.

FIGS. 195 A and B. — Parts of continuous tracing. Heart block, ectopic beats, auricular fibrillation (?). The ectopic beats are shown in the radial at r' r'' . Occasionally they are registered in the jugular only (c''); regularly recurring auricular beats are absent.

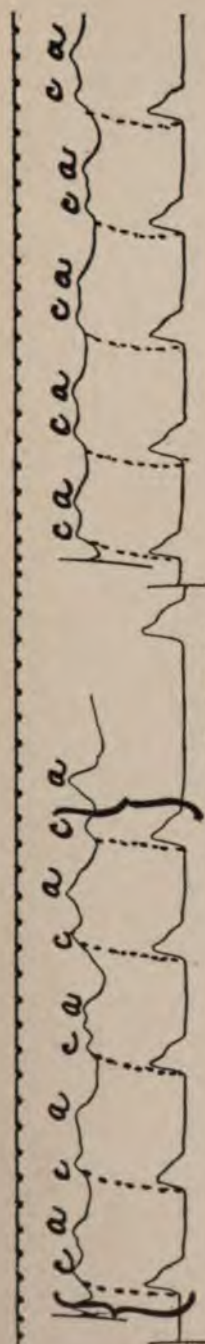


FIG. 196 A (1).

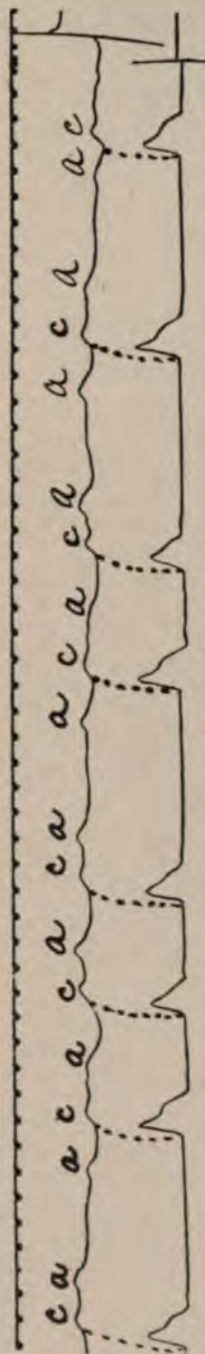


FIG. 196 A (2).

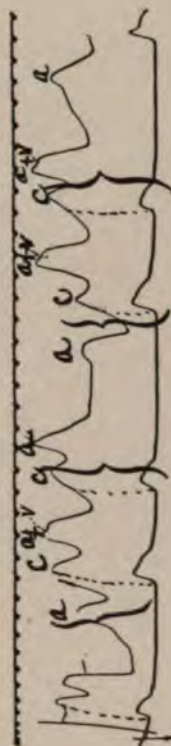


FIG. 196 B.

FIGS. 196 A and B. — Taken from the same patient on different days (1) and (2) are continuous). There are two ventricular rates: 66 and 47 per minute. The auricular speed is constant at 66 per minute. The conduction time (a-c interval) of the faster beats (within brackets) is extremely prolonged: from .5 to .55 second. The slower beats apparently occur when the conduction time is so prolonged that the ventricle no longer answers the auricular impulse and then beats at an approximately idio-ventricular rate.



Fig. 194. — Complete heart block with a ventricular extrasystole. The ventricular rate is 27; the auricular rate is 81 per minute. The pause following the extrasystole is not compensatory; it equals a rhythmic beat.

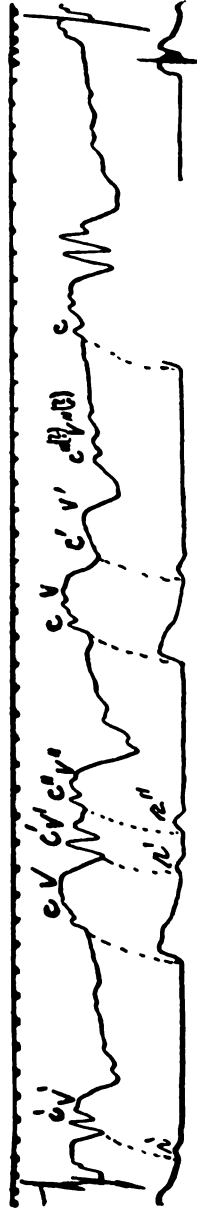


Fig. 195 A.



Fig. 195 B.

Figs. 195 A and B. — Parts of continuous tracing. Heart block, ectopic beats, auricular fibrillation (?). The ectopic beats are shown in the radial at r' r'' . Occasionally they are registered in the jugular only (c''); regularly recurring auricular beats are absent.

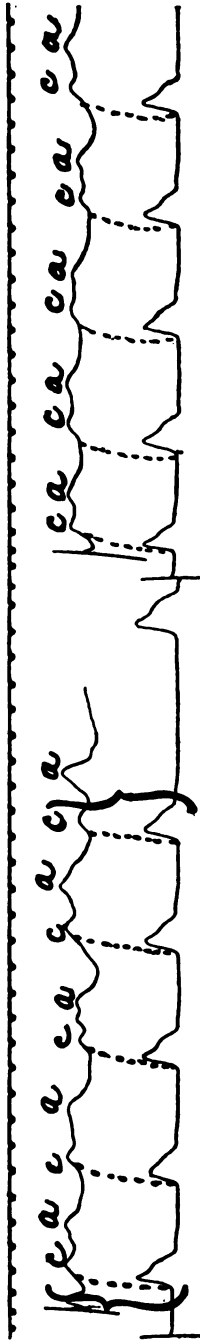


Fig. 196 A (1).

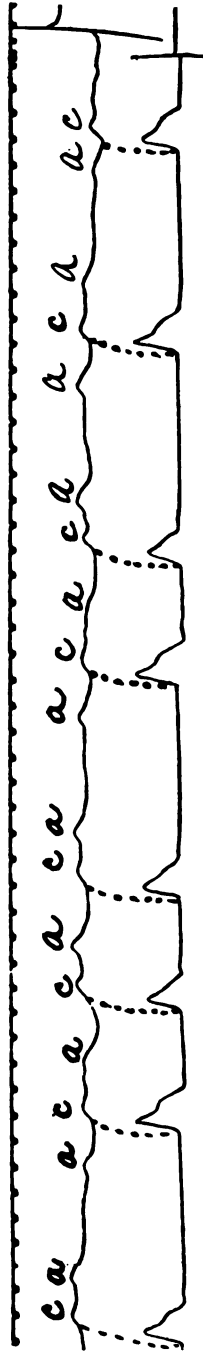


Fig. 196 A (2).



Fig. 196 B.

Figs. 196 A and B.—Taken from the same patient on different days (1) and (2) are continuous. There are two ventricular rates: 66 and 47 per minute. The auricular speed is constant at 66 per minute. The conduction time (a-c interval) of the faster beats (within brackets) is extremely prolonged: from .5 to .55 second. The slower beats apparently occur when the conduction time is so prolonged that the ventricle no longer answers the auricular impulse and then beats at an approximately idio-ventricular rate.

regular heart action. Polygraphically, the carotid (*c*) waves are identified in the usual manner. Since the auricular speed varies but slightly if at all, the auricular (*a*) waves are recognized by their occurrence at regularly spaced intervals (Figs. 187, 188). Because of their varying rates, the *a* and *c* waves occasionally fall simultaneously with, and are superimposed into, taller or broader peaks (Figs. 190, 193). Fig. 191 resembles incomplete block, because auricular and ventricular rates temporarily correspond. Examination of longer sections, however, show that this ratio is accidental, for, with slightly varying auricular and ventricular speeds, there is at first closer approximation and finally superposition of the *a* and *c* waves (Fig. 191*B*).

In complete heart block, auricular activity is occasionally abnormally rapid, so that its ratio to ventricular beats is as four or five to one. While regular rhythmic activity is the rule in complete heart block, there are exceptions; as, for instance, slight variations in the ventricular rhythm (Fig. 193). The regularity may also be disturbed by ventricular extrasystoles. These are usually single (Fig. 194, *r'*). The pause following them is not compensatory, but is equal in length to a rhythmic beat. Very rarely such extrasystoles are multiple (Figs. 195 *A* and *B*, *r'*, *r''*).

The case illustrated by Fig. 195 was originally one of complete heart block with a rhythmic ventricular rate of 35, and an auricular speed of 72 per minute, as demonstrated by polygraphic and electrocardiographic tracings. The auricular beats could be plainly heard in the interventricular pauses. Through inadvertence on the part of the nurse, digitalis was administered for several weeks after having been ordered discontinued. The pulse then became arrhythmic. The auricular beats were no longer heard. 'Extrasystoles' (i.e. ectopic beats), most of which were frustrane, were heard at the apex. The tracing shows some of them registered in both radial and jugular (Fig. 195 *A*, *B*, *r'*, *c'*, *r''*, *c''*); others in the jugular only (Fig. 195 *A*, *B*, *c''*); regularly recurring auricular waves could not be identified. Digitalis was discontinued. After two weeks, the original type of heart block was present, as shown by electrocardiographic tracings, and the auricular beats were again heard. From the auscultatory evidence, from the absence of auricular waves in the jugular, and from the fact that digitalis poisoning sometimes induces auricular fibrillation and 'extrasystoles,' it seems probable that the polygraphic tracing represents heart block, auricular fibrillation, and ectopic beats—a unique instance of digitalis poisoning in a patient with complete heart block.

Another rare example of peculiar disturbance of ventricular rhythm is that represented in Fig. 196 *A* and *B*, taken from a patient on successive days. Digitalis had not been given. The radial tracing showed ventricular arrhythmia due to abrupt and reciprocal changes from faster to slower rates. The faster rate was 66; the slower, 47 per minute. The auricular speed remained constant at 55. A study of the polygraphic tracing corresponding to the faster beats (included in the brackets) showed an extremely long conduction time, the *a-c* interval varying from .50 to .55 second. Occasionally the ventricle did not respond but contracted at the idioventricular rate (complete heart block). This condition existed for one week. Thereafter electrocardiograms taken frequently for a period of 1½ years always showed complete block (Fig. 179, Plate XV), the auricular speed being 100 to 110, the ventricular 45 to 50 per minute. The block was uninfluenced by atropin injections.

Higher rates are occasionally encountered in heart block. For example, in one of my cases, the ventricular rate was at one time as high as

PLATE XVI

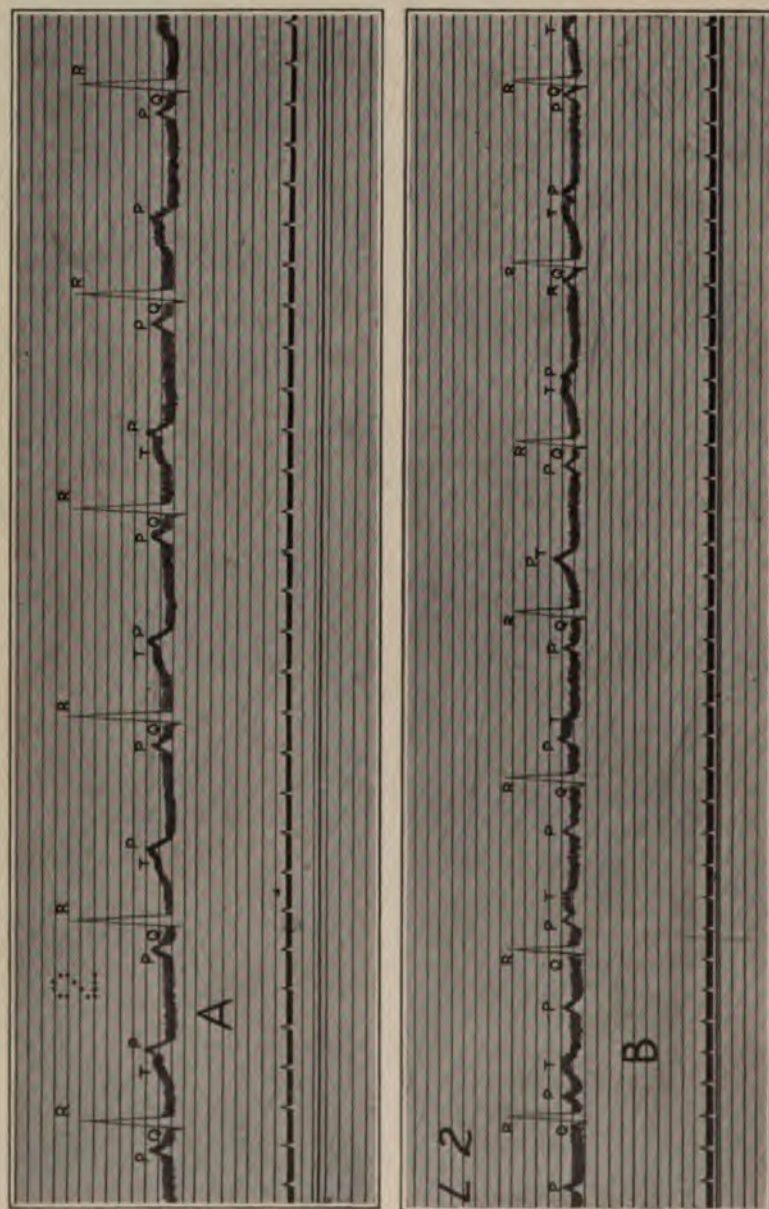


FIG. 198. — *A* and *B* are parts of the same electrocardiogram. *A* shows incomplete block with a 2:1 rhythm. *B* shows complete block.

77 per minute and the rhythm slightly irregular. The following are examples of heart block with varying auricular and ventricular rates. Figure 197 (Plate XV), from a man of 60 with myocarditis and aortitis, shows complete heart block. Figure 198 (Plate XVI) is given in two parts: *A*, illustrates incomplete block with a 2:1 rhythm; *B*, taken a few moments later, shows complete block. In section *A* the block only

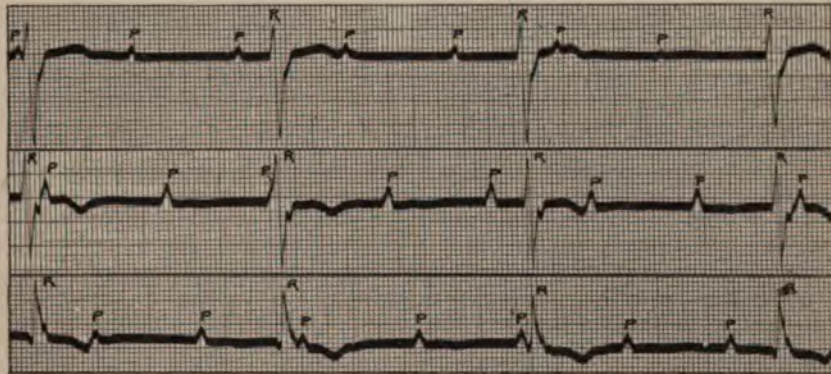


FIG. 197. — Complete heart block. The ventricular rate is 30; the auricular, 75 per minute. (Courtesy of Dr. A. E. Cohn.)

appears incomplete because of slightly varying auricular and ventricular speeds. This tracing was taken from a young man who had peritoneal tuberculosis with irregular fever and who, except for the arrhythmia, presented no sign of cardiac disease. The lungs were normal. The patient had been in the habit of feeling his pulse before his illness and had always found it of normal rapidity, that is, about 70 per minute. The peritoneal tuberculosis became quiescent following laparotomy. The heart block continued. Atropin was injected several times without influencing the block. Its only effect was a slight transient increase of the ventricular rate. The patient died two years later as the result of an accident. A necropsy was performed. In the abdomen, omental tuberculosis was found. The lungs were normal; there were no enlarged glands at the pulmonary hilus. On careful macroscopic examination, the cardiac valves and musculature were found normal. Though at the present writing, the atrio-ventricular conduction system has not been microscopically examined, there was no evidence of any gross lesion at the site of the bundle. In other words, there was no apparent pathological change in the heart itself to account for the block. This case will be etiologically discussed in another place (Chapter VIII).

Clinical Recognition of Heart Block. — The ventricular rate in complete heart block is usually between 25 and 40, although rates as

low as 8 per minute have been reported. The auricular beats are often heard in the apical region or at the third left interspace as soft faint distant sounds, interspersed between the ventricular contractions. Synchronous with the sounds of the auricular contractions, jugular pulsations unaccompanied by carotid beats may be seen in the neck. There are no compensatory pauses in the regular ventricular action of heart block, thus distinguishing this arrhythmia from extrasystoles. **Incomplete heart block** with changing ventricular ratios, for example from 2:1 to 3:1, requires differentiation from sino-auricular block; this distinction is based chiefly upon the auricular contractions which are present and sometimes audible in the first type of arrhythmia, and upon the absence of auricular beats, visible or audible, in sino-auricular block. It is sometimes possible to diagnose complete heart block merely from observation of the pulsations in the neck. Prominent pulsations indicative of the simultaneous contraction of auricle and ventricle ($a + c$ wave), or auricular with ventricular filling wave ($a + v$ wave) can then be observed. Similar large jugular waves sometimes seen with extrasystoles ($c'v'$, $a'c'$ or $c'a$ waves) are differentiated from those in heart block by the slow ventricular rate of the latter.

Pulse Alternation — Pulsus Alternans. — This is a disturbance, not of rhythm, but of **strength** of the **ventricular contractions**, and therefore of the pulse beats; strong beats regularly alternate with weaker ones. Alternation of the pulse is often not sufficiently marked to be detected by palpation (Figs. 199–200), hence the importance of radial tracings for its diagnosis. It occurs in tachycardia, especially when paroxysmal, and results then directly from the ventricular acceleration. Alternation is also fairly common after extrasystoles (Figs. 200–201).

This condition is usually found in patients with dangerously weakened myocardium and is commonly regarded as of grave prognostic import. However, alternation is occasionally found in patients with normal hearts, or as the result of digitalis medication. The question of the **mechanism** of **alternation** is the subject of many conflicting opinions and theories. The condition has been ascribed to disturbance of the function of contractility; to the fact that not all the ventricular fibers contract with the smaller beat; or because the systoles of the stronger are of longer duration than those of the weaker beats, thus encroaching upon the rest period of the latter. Electrocardiographic tracings of patients with alternation have partly upset some of these hypotheses. For example, there is no evidence that varying amounts of ventricular musculature are involved or that the path followed by the weaker differs from that of the stronger contractions; nor is there any difference in duration of their contraction times. Einthoven suggested an explanation similar to that involved in the irregular pulse excursions of auricular fibrillation, namely, that the increased blood pressure of

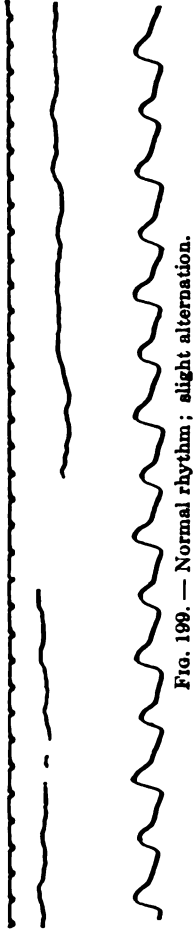


FIG. 199. — Normal rhythm; slight alternation.



FIG. 200.

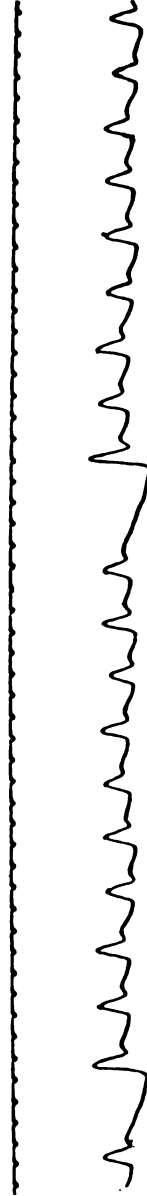


FIG. 201.

FIGS. 200, 201. — Alternation following extrasystoles.

the stronger contractions acting upon a weakened myocardium prevents the heart from properly emptying itself at the next systole, and consequently produces a smaller pulse. This theory puts the explanation a step nearer, but scarcely explains the *rhythmic* alternating regularity in pulse pressure.

Pulsus Paradoxus.—This irregularity is indicated by a gradual waning and waxing in the strength of the pulse beat with respiration, but without any change of rhythm. It has been regarded as characteristic of pericarditis with effusion. I have found it especially in cases of severe myocardial insufficiency. Occasionally the pulse becomes so weak and small as to be scarcely perceptible on palpation.

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CHAPTER VIII

THE ARRHYTHMIAS — THEIR ETIOLOGY AND THERAPY

At the outset it is important to emphasize that, although certain types of arrhythmias may be associated with various pathological entities, **almost any irregularity may be the result of non-organic, neurogenic, so-called 'functional' causes.** From the standpoint of etiology, therefore, it is necessary first to discover, if possible, whether the arrhythmia is of functional or of organic origin. Therapy will be only briefly touched upon here; fuller details will be found in the chapter on Circulatory Remedies (Chapter XVI).

SINUS ARRHYTHMIA

As a physiological phenomenon, sinus arrhythmia is quite common not only in children and young adults but in the middle-aged. It is, however, not confined to normal hearts. I have found the condition in young individuals suffering from aortal or mitral disease. The hearts of these patients were compensated; they had not received digitalis. Possibly this arrhythmia in patients with heart disease can be regarded as a favorable sign, since it is an evidence of physiological vagal control. It is also frequent as a phenomenon **after febrile respiratory catarrh** terminating by critical defervescence. The arrhythmia is then regarded by many observers as the result of toxic or infectious myocarditis. Its innocent nature, however, is proven by its disappearance within a few days, and by the absence of all signs of cardiac disease. Sinus slowing is sometimes a purely pathological occurrence; it then bears no relation to breathing. For example, it occurs in severe secondary anemia following sharp hemorrhage. I have observed two such cases. In both the sensorium was clear and the arrhythmia gradually disappeared with improvement in the anemia. In such instances, I believe sinus arrhythmia due to nutritional changes in the sino-audicular node, or to increased vagus inhibition on the part of the cardio-inhibitory center. In another case, an adult whose heart at necropsy showed marked presenile cardiosclerosis involving the endocardium, mitral valves, aorta and the coronaries, the pulse ranged between 50 and 60

during the last year of life. Polygraphic tracings showed sinus arrhythmia. Microscopical examination of the sino-auricular node was not made; sclerotic changes in that region may have been the etiological factor.

Physiological sinus arrhythmia, or sinus arrhythmia encountered in febrile crises, requires no medication. When due to the other etiological factors above mentioned, treatment should be directed to them rather than to the arrhythmia itself.

SINO-AURICULAR BLOCK

This is usually the result of digitalis poisoning. It has also been produced by pressure over the vagus in the neck, by pressure upon the eyeballs (oculo-cardiac reflex) in human beings and by various experimental procedures in animals. It is occasionally the result of tobacco poisoning (Fig. 178, Fig. 179, Plate XV). Otherwise it is exceedingly rare as a clinical phenomenon. The condition in itself requires no medication unless accompanied by dizziness; atropin sulphate is then sometimes of value.

EXTRASYSTOLES

Though there are exceptions, auricular extrasystoles are usually of functional origin; the ventricular may be functional or organic. When organic, auricular or ventricular extrasystoles may mark the beginning of decompensation and then remain as permanent arrhythmias. In acute heart failure, they are sometimes the forerunners of a fatal termination. The onset or exacerbations of acute endo- or pericarditis may be accompanied by extrasystoles. We do not know in what manner organic lesions increase ventricular excitability and thus produce premature contractions, although it seems probable that impaired cardiac nutrition plays an important rôle when severe cardiac disease is present.

When extrasystoles are found in cardiac failure, the treatment of the latter is the prime consideration (Chapter XVI). When caused by endo- or pericarditis, the therapy consists mainly of salicylates in large doses. Occasionally the addition of bromides is of value.

Functional Extrasystoles of Extracardiac Origin. — **Cerebellar and cerebral** conditions which exert pressure directly or indirectly upon the cardio-inhibitory center can cause premature contractions. These factors apparently act by direct excitation of the vagus center. Thus, a patient with cerebellar tumor and normal circulatory apparatus had occasional extrasystoles during the last few months of life. That they were due to pressure was demonstrated at operation for removal of the tumor. The latter was deeply seated, each attempt at its removal being accompanied by premature contractions. **Acute gastric disease** with vago-excitative characteristics, less often **intestinal disease**, are occasionally accompanied by extrasystoles, usually auricular in type.

Their etiology has been variously explained. The usual theory is that, after a meal, the distended stomach presses the diaphragm against the heart, thus embarrassing circulation. Another reason given is the action of absorbed toxic products upon the neurogenic cardiac control. In the majority of cases that have come under my observation, the extrasystoles occurred most frequently when the stomach was empty. Several of these patients, fluoroscoped after a bismuth meal, showed no gastric distention or other evidence of abnormal pressure against the diaphragm; in fact, their stomachs were contracted and hypermotility was present. These observations nullify, I believe, the commonly advanced theories. The intimate physiological relationship between the nerves of the stomach and those of the heart lead me to believe that extrasystoles accompanying gastric disturbances are probably caused by reflex excitation of the cardiac nerves. The path of the reflex arc is not clear.

Acute inflammation of the gall-bladder and bile ducts, with or without gastric symptoms, may also be accompanied by premature contractions. Though the theory is based upon insufficient data, toxemia has been assumed as the etiological factor for the extrasystoles. The arrhythmia is often present very early in the disease, when extensive toxic absorption appears improbable. Similar to the views expressed with reference to extrasystoles in gastric disturbances, I believe that most cases occurring in gall-bladder disease are caused by reflex excitation of the neurogenic control of the heart, though at present the centripetal nerve-path involved is unknown.

Peritonitis is another disease in which premature contractions may occur. One case of appendicitis, followed by general peritonitis and death, in an elderly individual with cardiosclerosis, had ventricular extrasystoles at the onset of the attack; during the last few days of life, auricular fibrillation was present. It was impossible to decipher the varying rôles played by toxemia and reflex nerve excitation in the production of these arrhythmias; in this case I believe both were factors.

The **onset and crisis of acute catarrhal febrile affections** are frequently marked by premature contractions. The latter are generally regarded as evidence of toxic myocarditis. For several reasons this relationship seems doubtful to me. Most of the cases I have observed were not toxic: the extrasystoles occurred with mild grippe and tonsillar affections in patients who scarcely felt ill. The arrhythmia was found at the crisis or immediately thereafter when the severe symptoms had disappeared. There were no circulatory symptoms. On clinical grounds, therefore, it seems probable to me that these extrasystoles are due to abnormal products (toxins?) flowing in the general circulation and affecting the **cardio-inhibitory center** and not the myocardium.

Regarding **extrasystoles in pneumonia**, it should at the outset be stated that this disease can insidiously cause severe cardiosclerosis (Chapter XI); but this does not apply to the question of premature contractions occurring in the **acute stage** of pneumonia. Unless cir-

culatory symptoms are already present, pneumonic patients with critical or post-critical extrasystoles do not suffer from cardiac failure as a result of the arrhythmia. The latter usually disappears within a few days without any treatment. If myocarditis or other severe pathological damage in the heart caused these extrasystoles, symptoms of circulatory failure, instead of being absent, would be prominent. The present theory regarding crises is that the system is at such times suddenly flooded with toxins elaborated in the pneumonic area. That the cerebral centers become involved is shown by critical sweats and vasomotor symptoms. Correlating these observations, it seems most likely that the extrasystoles are the result of pneumonic toxins acting on the cardio-inhibitory center.

Among drugs, the **digitalis** bodies, less frequently the **salicylates**, produce extrasystoles. When the former are given, the arrhythmia is usually coincident with their full therapeutic effect (Chapter XVI). Fright and epileptic seizures, overindulgence in coffee, tea, and tobacco, are additional causes of premature contractions. The latter also occur in conjunction with the vasomotor symptoms of the climacterium.

There still remains a group of patients in whom no cause for extrasystoles can be discovered. It is interesting in this connection to note that functional extrasystoles, even though they later disappear, are apt to recur after any slight disturbance (for example, overexertion, acute indigestion) which reflexly affects the normal inhibitory control.

Therapy.—Such underlying diseases as gastric disturbances, appendicitis, etc., naturally require their appropriate remedies; the presence of extrasystoles in no wise affects the usual therapeutic indications and procedures. Unless extrasystoles of reflex or neurogenic origin cause such subjective sensations as “fluttering in the chest,” transient faintness, etc., or are in themselves causes of circulatory failure (an exceedingly rare occurrence), they require no therapy. When medication is indicated, the bromides are of most value. Digitalis has occasionally been advised because of its effect in increasing vagus inhibition.

TRUE BRADYCARDIA

The term has already been defined (Chapter VII). It may appear as a rare congenital anomaly in patients with normal hearts and circulation. A sequential rhythm and pulse rate between 45 and 60 per minute are sometimes encountered in senile cardiosclerosis. Most cases of true bradycardia are of **extracardiac** origin. Patients with **lead colic** and **gastro-intestinal disorders**, accompanied by abdominal pain, belching, or diarrhea, are apt to have a slow pulse. This is probably due to reflex vagus inhibition from excitation of gastric and intestinal nerves.

Catarrhal jaundice is not infrequently accompanied by true bradycardia, and is usually ascribed to toxic myocarditis. In the patients whom I have observed, there was no evidence of the latter disease.

The bradycardia in these cases may be ascribed to two causes: in those with cholemic symptoms it is probably of central inhibitory origin; in non-toxic cases with painful local symptoms, it is due, I believe, to reflex vagus inhibition from excitation of the nerve filaments surrounding the gall bladder.

Appendicitis. — Slight bradycardia with pulse rates between 60 and 65 per minute is occasionally encountered in appendicitis. Myocardial involvement or quiescence of the inflammatory lesion has usually been assumed as the cause. Unless general peritonitis and severe toxemia are present, the first factor seems improbable. Regarding the second factor it appears more likely that a slow pulse is an indication of continued and active excitation at the inflammatory focus rather than an evidence of its quiescence.

Among **drugs**, the digitalis bodies and salicylates cause this arrhythmia by increasing vagal inhibition.

Epileptics occasionally have an abnormally slow pulse. This is also true of those types with so-called vago-vasal attacks (Gowers) in which there are no convulsions, but vasomotor symptoms, vomiting, syncope, and pains along the distribution of the intercostal nerves.

A slow pulse is also fairly common at the **crises of febrile pulmonary affections**, or during the course of the disease. When present at the crises, I believe it is due to the neurotropic effect of the toxins liberated during critical defervescence.

Therapy for bradycardia is only indicated when the slow pulse rate is accompanied by subjective symptoms. Atropine is then of most value, given first in small, later in increasing doses, until the full physiological effect has been reached.

CLINICAL AND EXPERIMENTAL CAUSES OF AURICULO-VENTRICULAR HEART BLOCK

- | | | | |
|-----------------------|---|---|---|
| EXTRA-CARDIAC FACTORS | I. Cardiac Causes | { | (a) Destruction of the A-V bundle or auriculo-nodal junction by tumors (benign or malignant), calcareous deposits, fatty and inflammatory infiltration, fatty degeneration, coronary infarct, hemorrhage in the node, fibrosis. |
| | | { | (b) Severe myocardial degeneration. — Destruction of the branches or terminal arborizations of the conduction system. |
| | II. Drugs (Digitalis — Morphine — Nicotine(?)). | | |
| | III. (a) Asphyxia. | | |
| | (b) Chemical Poisons | { | producing acidosis.
toxins.
anaphylotoxins. |
| | IV. Interference with cerebral circulation. | | |
| | V. Abnormal pressure on the cardio-inhibitory center. | | |
| | VI. Abnormal pressure on the vagus. | | |
| | VII. Increased vagal inhibition. | | |
| | (a) Digital pressure on the vagus. | | |
| | (b) Peripheral excitation. | | |

Heart block is here used to denote auriculo-ventricular dissociation, complete or incomplete; it does not refer to lengthened conduction time, blocked auricular beats, or to sino-auricular block. I have made and adopted the etiological classification given above, which probably includes the great majority of known factors.

I. (a) Any lesion which completely severs or destroys the main branch of the conduction system produces complete heart block. If the destruction is incomplete, there may be enough healthy strands to carry impulses from auricle to ventricle. A suddenly deficient coronary supply, the result of thrombosis, infarct, or disease of the nutrient artery of the bundle of His, can so profoundly disturb the nutrition of the conduction system as to produce transient or permanent heart block.

I. (b) Just as fibrotic and calcareous degeneration in the conduction system are almost invariably accompanied by gross pathological changes in the ventricular musculature, so in extensive cardiosclerosis there are often fibrotic and other degenerative changes in the connecting bundle. Even if the bundle is sufficiently normal to convey auricular impulses, cardiosclerosis in itself can probably produce heart block by the destruction of the branches or terminal arborizations of the conduction system in the ventricular musculature, in this manner preventing impulses from reaching their final destination. On the other hand, even if these branches and their ramifications are sufficiently normal to carry impulses from auricle to ventricle, the ventricular musculature may be so diseased as not to be capable of a normal response. Any or all of these causes (group I (b)) may operate in those cases of heart block in which the main conduction system is found quite or fairly normal upon pathological examination.

Therapy. — When syphilis is the etiologic factor, vigorous antiluetic treatment — salvarsan, bichloride injections, and iodide of potash are indicated (Chapter XIV). The advisability of using digitalis and atropine in cardiac disease with heart block is discussed in a subsequent chapter (Chapter XVI).

II. **Digitalis** can produce complete or incomplete block. The arrhythmia has been observed not only in those patients with heart disease, but it has also been experimentally induced in those with healthy hearts. Complete heart block has been induced by the injection of **morphine** in dogs, and was there regarded as an effect upon the cardio-inhibitory center. Morphine poisoning in man is sometimes accompanied by very slow pulse rates. These have never been graphically recorded; they may be due to the same cause found in the animal experiments; namely, heart block. **Tobacco poisoning** is occasionally accompanied by a very slow pulse. To determine its precise nature, graphic tracings are necessary. In this manner it may in the future be demonstrated that some of these tobacco arrhythmias are likewise due to heart block.

III. (a) In **animals**, the experimental production of **asphyxia** is

accompanied by varying degrees of heart block; the essential cause is assumed to be nutritional disturbances from lack of oxygen in the junctional tissues. With our present imperfect knowledge of the chemistry of the blood, it is impossible to state whether **other chemical factors** (group III (b)) can act similarly in the production of heart block by profoundly interfering with cardiac nutrition. Such factors are: abnormal constituents in the blood, causing lessened blood alkalinity (acidosis); chemical poisons elaborated in infectious diseases (**toxins**); and **anaphylotoxins**. Regarding acidosis, I have several times observed heart block in the agonal stages of patients suffering from cardiosclerosis and nephritis, in whom electrocardiograms showed very rapid and irregular auricular and ventricular activity (Agonal Arrhythmias, Chapter VII). Cyanosis was not a constant factor. Blood examinations for non-protein nitrogen and other products were not made, but from the clinical syndrome, it seemed that retained chemical poisons were the essential cause of the arrhythmia. With reference to **toxins**, it has been shown experimentally that transfused pneumonic blood profoundly impairs cardiac contractility. I have had occasion to study two cases which have a bearing upon these experimental observations. Both developed heart block during the course of pneumonia. In one an autopsy was performed. Upon macroscopic examination the heart was found normal. Careful microscopical examination of the **conduction system** also showed that it was normal. Such clinical and experimental observations indicate that in pneumonia, at any rate, toxins have not only an action upon the cerebral centers, but also a local action upon the heart. Block may then result from interference with nutrition of the *A-V* conduction system either alone or in conjunction with the remainder of the cardiac musculature. Complete heart block has been produced in animals by producing anaphylaxis; the cause is assumed to be due to an effect upon the heart itself.

IV. Interference with Cerebral Circulation.—This factor probably operates by interference with nutrition of the cardio-inhibitory center. Sclerotic changes in the arteries at the base of the brain is the pathological condition most frequently observed. In one reported case of heart block, few changes were found in the conduction system or cardiac musculature, the cerebral arteries constituting the circle of Willis were markedly diseased, the brain was normal. Tumors and other pathological conditions which impede cerebral circulation may have a similar effect in producing heart block.

V. Abnormal Pressure on the Cardio-inhibitory Center.—Such pressure is directly or indirectly brought about by cerebral, cerebellar or pontine tumors, ventricular cysts, meningitis, hydrocephalus, etc.

VI. Abnormal Pressure on the Vagus.—Tumors and adhesions are the most likely pathological entities which may have this effect. One case of heart block from pressure on the vagus has been reported; a large tumor in the anterior mediastinum involving the nerve was found.

VII. Increased Vagal Inhibition. — It has been shown that in children with heart disease, digital pressure on the vagus in the carotid sheath may be followed by temporary heart block. This manœuvre probably acts by causing increased vagal inhibition. A case has also been reported in which swallowing induced heart block in a patient with delayed conduction time. These are examples of the induction of this arrhythmia resulting from direct excitation of the vagus in susceptible individuals.

(b) **Reflex Peripheral Excitation of the Vagus.** — I have observed cardiac inhibition with the production of block, apparently reflexly invoked by peripheral stimulation of the pneumogastric branches supplying the stomach. The clinical history of the patient showed that the pulse had been normal before the onset of tuberculous peritonitis from which the patient suffered when he first came under observation. For several weeks high temperature and abdominal pain had been present. The abdomen was opened, and fluid evacuated. Some weeks after operation, the symptoms of tuberculous peritonitis entirely subsided. Two years later the patient was killed in an accident. Complete heart block, as shown by polygraphic and electrocardiographic curves, was present before and after the operation, and up to the time of the patient's death. The presence of the arrhythmia caused no symptoms. A necropsy was performed; the lungs were found normal; a mass of enlarged non-suppurating tuberculous glands adherent to stomach and intestines was found. Careful macroscopic examination showed the heart to be absolutely normal. At the present time the microscopical examination has not been completed; but careful scrutiny of the conduction system showed the absence of any gross lesion. Toxemia as a cause of the heart block could be excluded, for the patient was clinically well, had had no temperature, and was at work for months prior to his death. Weighing the pathological and clinical data, it seems probable to me that the heart block was due to abnormally increased vagal inhibition reflexly excited by involvement of peripheral nerve filaments in the tuberculous foci.

Therapeutically atropine sulphate should be tried in all cases of heart block; it should be given hypodermically until the full physiological effect is reached. Appropriate treatment should be directed against the individual etiological factors.

PROLONGED CONDUCTION TIME

Among drugs, digitalis occasionally produces this arrhythmia. The chief cardiac cause is myocardial degeneration; indeed, a prolonged *a-c* interval may be the only evidence of this disease. Prolonged conduction time is encountered during the course of, or immediately after, attacks of rheumatic endocarditis. It is also met with in acute or sub-

acute nephritis in which there are none of the usual clinical manifestations of myocarditis. The arrhythmia is occasionally of neurogenic origin. **Therapy** is indicated for the symptoms of myocarditis or for other etiological factors, but not for the prolonged conduction time.

SHORTENED CONDUCTION TIME

I have found this arrhythmia especially frequent in exophthalmic goiter (Chapter IV). The fact that experimental excitation of the right accelerator nerve is accompanied by a slightly, and of the left, by a considerably, shortened conduction time is doubtless of etiological significance in exophthalmic goiter, in which the chief symptoms are due to excitation of the sympathetic system. With shortened conduction time tachycardia is usually present; the latter with other clinical manifestations may require medication, the shortened conduction time in itself does not.

AURICULAR FIBRILLATION

This is the usual arrhythmia accompanying decompensation in rheumatic mitral stenosis in the young and middle-aged. It is sometimes found in children; I have seen several instances in patients between 10 and 12 years of age. The frequent association of auricular fibrillation with mitral stenosis has never been satisfactorily explained. It is known that stenosis is often accompanied by hypertrophy and dilatation of one or both auricles. Destructive changes in the sino-auricular node, similar to those observed in older patients with cardio-sclerosis and auricular fibrillation, have been described in isolated instances. In addition to these profound pathological changes, it appears to me that mechanical auricular overdilatation (to which attention has not been directed) may play an etiological rôle in the production of the arrhythmia. Overdilatation may, for example, prevent the proper performance of coördinate rhythmical contractions to normal stimuli, and in their stead, the answer to stimulus production consists in the irregular, vermicular, incoördinate contractions characteristic of auricular fibrillation. The auricular structure and the method of transmission of the excitation wave (Chapter VI) explain fibrillation in both auricles after one has been affected.

Auricular fibrillation is found less frequently in rheumatic aortal lesions. It is an extremely common arrhythmia in all types of cardiovascular disease in the aged (Chapter XV).

In cardiac disease with decompensation auricular fibrillation is usually permanent. It may, however, be transient or occur only in attacks. I have observed such attacks lasting several days in two cases of aortic aneurism. In one, it occurred with the gradual onset of severe de-

compensation; in the other, there was no discoverable cause for the attacks. Another instance of transient auricular fibrillation was that of a woman of 60, a sufferer from mild myocardial insufficiency, who had been operated upon for empyema of the gall-bladder. Following the operation, there were several distinct attacks of broncho-pneumonia, the onset of each being marked by a moderate rise of temperature, and by auricular fibrillation lasting one day. In mitral stenosis, *pari passu* with fresh exacerbations of endocarditis, auricular fibrillation may occur. Thus, in a man of 45 with a double mitral lesion, from whose blood a non-hæmolytic streptococcus was isolated, each sharp febrile invasion was accompanied by an attack of auricular fibrillation; these attacks lasted several hours or days. Patients with mitral disease who suffer from acute febrile disturbances of non-rheumatic origin are also prone to attacks of this arrhythmia; these may last throughout the fever. I have seen two such instances: one, a patient with mitral regurgitation, the other with mitral stenosis; both developed fibrillation during erysipelas. Auricular fibrillation may also be an initial symptom of coronary embolism or thrombosis.

From these observations it is evident that in diseased hearts any additional insult to the endocardium, myocardium, or coronary arteries may be accompanied by auricular fibrillation. The permanence of the arrhythmia may depend upon the severity or permanence of the pathological damage.

The sovereign **remedy** for this irregularity when found in decompensated cardiac disease is digitalis. The details of its administration are elsewhere described (Chapter XVI). I have occasionally added large doses of bromide, or have initiated digitalis medication with one or two large doses of morphine when patients were very dyspnoic or restless.

Auricular fibrillation occurs very exceptionally in patients with normal hearts. I have observed several cases of temporary auricular fibrillation coming on at the critical defervescence of lobar pneumonia; there was no evidence of heart disease or heart failure during the entire course of the disease or during convalescence. Some of these patients were examined months after the pneumonia. Their hearts were found perfectly normal. Temporary or permanent auricular fibrillation is also fairly common in exophthalmic goiter. Apparently here influences affecting neurogenic cardiac control are the essential factors. I have likewise seen auricular fibrillation in a man with a normal heart who was a very heavy smoker. A case due to hydrogen sulphide poisoning in a man with a normal heart has recently been reported.

In those cases of fibrillation which are apparently of toxic or of neurogenic origin, I have found very little value in digitalis; the pulse or ventricular rate was not appreciably affected; dyspnoea, if present, continued. Large doses of bromide, alone or combined with small doses of codeine, seemed occasionally of value.

SIMPLE TACHYCARDIA — ORDINARY PULSE ACCELERATION

Rapid, regular pulse rates usually range between 120 and 180 per minute, and are the result of excitation of the accelerator nerves. The causes are manifold. Excitement, fright, overexertion, fever are some of the commoner. In exophthalmic goiter, tachycardia is one of the cardinal symptoms. Gastro-intestinal disturbances of functional or organic nature are frequently accompanied by rapid heart action. Persistent tachycardia may be the only clinical evidence of a fresh exacerbation of an old endocarditis. Cardiac decompensation from any source is often accompanied by moderate tachycardia, and is sometimes the forerunner of other arrhythmias. Among other causes of rapid heart action are dyspnoea from pulmonary disease, tabagism, overindulgence in tea and coffee, and atropine.

Pulse acceleration requires medication only when the pulse rate is high or subjective sensations are present. When tachycardia is due to fever, digitalis has no effect in reducing the pulse rate. The bromides or codeine are occasionally efficacious in slowing the heart in such cases. Digitalis may decrease the pulse rate when the tachycardia is due to cardiac decompensation.

PAROXYSMAL TACHYCARDIA

This is more often of functional than of organic origin. Its most frequent extracardiac cause is acute indigestion, and is then apparently due to reflex excitation of the accelerators. I have seen several patients in whom every attack of indigestion was accompanied by this arrhythmia. In a woman of 50 with abdominal cancer, gastric symptoms and severe secondary anaemia, slight fright or nervous excitement would frequently initiate an attack. At necropsy, the cardiac musculature was somewhat pale, otherwise the heart was normal. In another patient, a woman of 45 with a luetic history and positive blood Wassermann, sudden gastric attacks also initiated paroxysmal tachycardia. It is interesting to note that during these attacks there was marked dilatation of the left pupil, an evidence of sympathetic nerve excitation.

In addition to **treatment** directed to the cause (gastric sedatives, etc.), sudden firm pressure over the vagus in its course in the neck may occasionally suddenly arrest the arrhythmia. Pressure on the right side should be first tried; if this does not succeed, pressure should be exerted over the left vagus. Similarly, pressure on the right or left eyeball can be tried in the attempt to induce the oculo-cardiac reflex. Inducing emesis may also affect the vagus control. Strophanthin may be employed hypodermically or intravenously because of its quick, powerful effect in increasing vagus inhibition. If these procedures or drugs are unsuccessful, morphine or the bromides are indicated.

AURICULAR FLUTTER

This arrhythmia is most frequent in older people with cardiosclerosis in the period of decompensation. It can also occur during the course of acute endocarditis without decompensation. Occasionally auricular flutter is of functional origin. In acute endocarditis, digitalis does not control the arrhythmia. Since the drug has not been employed in flutter of functional nature, nothing is known of its effect in such cases. In auricular flutter with decompensation and cardiosclerosis, digitalis (Chapter XVI) is of very great value. Its administration is then followed by auricular fibrillation; if the drug is then discontinued, normal rhythm is resumed, and, coincidentally, return to compensation.

VENTRICULAR ESCAPE — INDEPENDENT VENTRICULAR ACTIVITY

A brief report of a case of this interesting and rare arrhythmia follows. The etiological factors involved in this and other patients with this arrhythmia is discussed in the comment following the case report.

S. P., male, aged 20, was first observed on February 4, 1913. He had measles when 3 years old and typhoid when 10; otherwise there was no history of any previous illness. He was not addicted to tea, coffee, tobacco, or alcohol. Five days previously he developed a typical attack of acute articular rheumatism involving the ankles, wrists, knees, and elbows. The attack was accompanied by fever; there were no chills or gastric disturbances.

Except for swelling and redness of the inflamed joints, the general and neurological examination revealed nothing abnormal. There was no urethral discharge. The complement-fixation test for gonorrhœa was negative. The cardiac outline was normal to percussion, the apex beat was in the fifth interspace, 8.5 cm. from the midsternal line; the heart sounds were normal; the pulse was rhythmical. The systolic and diastolic blood pressures were within normal limits. The temperature ranged between 101° and 103°. There was no dyspnœa. The patient did not appear very ill. Sodium salicylate in moderate doses was given for two days.

Two days after hospital admission, a transient pulse irregularity appeared. Six days thereafter, it reoccurred and clinically resembled extrasystoles; no tracings were made at that time. February 12, the irregularity occurred every third or fourth beat. From that day frequent polygraphic and, later, electrocardiographic tracings were taken. February 14, for the first time a rough blowing systolic murmur was heard at the apex. Occasionally there were runs of from three to twelve stronger thumping beats unaccompanied by the murmur; studies of the tracings showed that these beats were due to simultaneous action of auricle and ventricle. Four days later the arrhythmia appeared only

infrequently and the systolic murmur had almost entirely disappeared. The patient left the hospital feeling well.

As possible **causes** for the production of automatic ventricular action, neurogenic, toxic, and organic factors require consideration. A neurogenic factor in the sense of a so-called neurosis due to extracardial conditions (for example, gastric disorders) causing abnormal excitation in the centripetal arm of a reflex arc, can be here dismissed because of the type of the disease, its course, and the definite completion of the arrhythmia with the end of the rheumatic attack. Concerning toxins their action seems to depend upon their complicated chemical composition and upon intricate chemical reactions taking place in the body. By analogy, it seems possible that a rheumatic toxin may also produce a similar arrhythmia, though there is no evidence for the assumption in this case. Concerning an organic cause for the arrhythmia, it is recalled that the patient developed a loud systolic murmur at the apex, one week after the appearance of the arrhythmia; the murmur remained for two days, then gradually disappeared. It also disappeared when auricle and ventricle contracted simultaneously, an apparent corroboration that it was due to mitral insufficiency, organic or relative in nature. It is not my intention to discuss cardiac murmurs at any length in this connection. Briefly, systolic apical murmurs which occur during the course of any febrile disease, and then disappear without evidence of an organic cardiac lesion, are by no means infrequent. On the other hand, organic murmurs usually increase in intensity and do not disappear. The occurrence of the murmur in conjunction with acute articular rheumatism makes its presence suspicious of some transient valvular or myocardial involvement. Rheumatic infections cause myocardial inflammation in the form of submiliary myocardial nodules (Aschoff bodies, Chapter XII). Healed or healing isolated Aschoff bodies have been found on the interventricular septum in hearts which were the subjects of rheumatic reinfection. During their inflammatory state, if situated close to or even partly involving the bundle of His before its division, they can conceivably cause local irritation sufficient to produce occasional ventricular automatism with beats of supraventricular origin. The bundle, however, need not be sufficiently compromised to prevent the idioventricular impulse from following its normal course in the conduction system — a fact which probably accounts for identical electrocardiographic complexes of all beats, rhythmic and arrhythmic.

Right and left vagus pressure had no effect on auriculo-ventricular sequence. One of the atropine experiments was followed by a number of independent ventricular contractions, with no marked difference between ventricular and auricular rates. This observation does not necessarily exclude the possibility of an organic cause for ventricular automatism, because an irritative lesion which does not entirely and permanently compromise the bundle may upset normal nerve control

and mechanism and make the latter susceptible to atropine poisoning. It would thus seem that the automatic ventricular mechanism was not sufficiently sensitive to respond to vagus pressure, but that atropine poisoning prevented the inhibitory vagus control and permitted ventricular escape.

Summarized, a case of independent ventricular activity is described; the lowest ventricular rate is 56 per minute, the usual rate is 60 and remains so whether ventricular automatism is present or not. The electrocardiographic complexes of all beats are identical. At one time atropine administration is followed by ventricular escape. The occurrence of automatic activity during the course of acute articular rheumatism and its disappearance later, and a study of the physical signs make it probable that a small transient myocardial inflammatory focus at or near the auriculo-ventricular bundle is the irritative cause of the abnormal mechanism.

Therefore it may be stated that transient independent ventricular activity may occur with no change in the path followed by the idio-ventricular impulse, with no difference of rate between normal and abnormal beats, and with no marked retardation of the auricular rate.

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CHAPTER VIII

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CHAPTER IX

ORTHODIASCOPY AND FLUOROSCOPY

Definition of Orthodiascopy. — Orthodiascopic examination consists essentially in a method of outlining a viscus by means of parallel roentgenographic rays; in this manner the exact size of the organ is reproduced. With the tube stationary and near the patient, the rays are divergent; hence the object to be examined becomes artificially enlarged. This distortion is magnified if the organ itself be large, for the impinging rays become still more divergent; an hypertrophied heart, for example, produces a disproportionately enlarged shadow. This was well exemplified in a case in which the roentgenogram taken two days before death showed the shadow of a tremendously enlarged heart. At necropsy the heart was found only moderately enlarged; pericarditis causing a tightly adherent pericardium prevented agonal cardiac dilatation which might have been assumed as the cause for the enlarged roentgenographic shadow.

Two methods of avoiding distorted images have been devised.

(1) **Teleroentgenography**: the X-ray tube is placed about 6 feet from the patient, so that the rays reach the organ approximately parallel. This method has given satisfactory and fairly accurate results. (2) **Orthodiascopy**: the focus of the X-ray tube and the center of the screen are adjusted so as to be in a straight line; the X-ray tube and the fluoroscopic screen move together. Any series of points which outline a viscus are brought in alignment so that, with the movement of the tube and screen, parallel and not divergent rays impinge upon the organ and reach the observer.

ORTHODIASCOPY

The **Groedel apparatus** is the one I have found most suitable for orthodiascopy of the heart. Examinations can be made in the sitting, standing, or lying postures. For convenience, and the comfort of the patient, I prefer the sitting position. After the patient is properly seated (see legend, Fig. 202, Plate XVII), the room darkened, and the electric foot switch turned on, a rapid survey is first taken of the entire

PLATE XVII

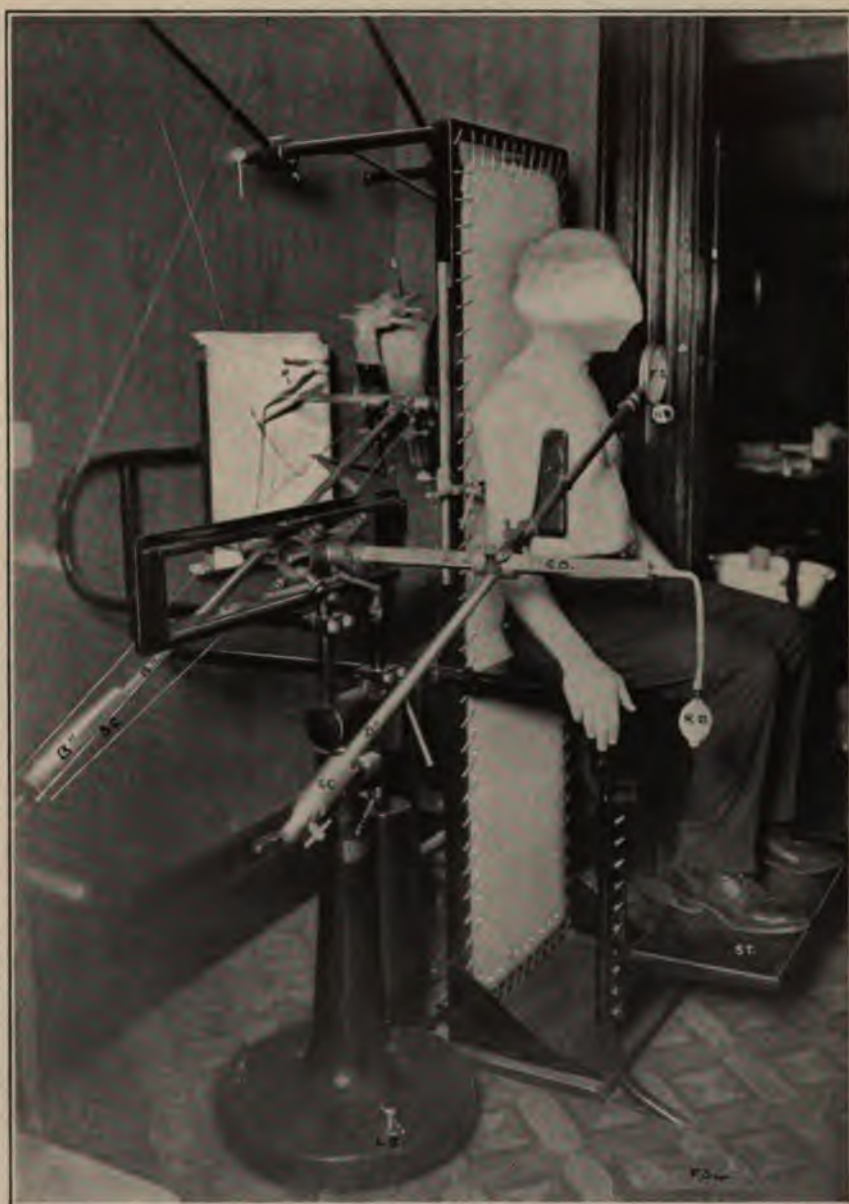


FIG. 202. — Groedel Orthodiascope, front view.

The patient sits with his feet resting comfortably on the adjustable step (*ST*); the side plates (*SP*) can be lowered or raised and pushed in or out; they are placed so that they fit snugly in the axillæ and against the chest of the patient. The fluoroscopic screen (*FS*) is grasped by the finger of the right hand in a small ring (*R*). The bar *B'* carrying the screen is connected by the hollow crossbar (*CB*) with the bar *B''*; the latter carries the X-ray tube so that any motion imparted to the fluoroscopic screen by the observer moves the X-ray tube as well. By loosening the nut (*N*) the bar (*B'*) may be slid along the crossbar and the screen may be placed any convenient distance from the patient's chest.

RB = rubber bulb used for marking purposes;

SC = sliding counterweights so that the screen and X-ray bars may be properly counterpoised;

C = cranks used to place the table in the horizontal position if necessary;

LS = leveling screws;

FSw = the electrical foot switch.

PLATE XVIII



FIG. 203. — Details of marking mechanism: XR = X-Ray tube which may be raised or lowered in its frame (F). It is partially protected by the rubber shield (RS). The marker (M) is in line with the center of the screen (see Fig. 202) and the focus of the X-ray tube. The rubber tubing ($R.T$) is connected with the rubber bulb RB (Fig. 202) through the hollow cross bar. When the bulb is squeezed and the marker inked, the pencil is pushed forward and dots the paper (P), fastened upon the board (B). W are the wires connecting the X-ray tube with the coil.

heart by moving the fluoroscopic screen over the cardiac area. The cardiac outline is then systematically mapped out. The central spot of the screen (*F.S.* Fig. 202, Plate XVII) is brought over the edge of the area to be delimited; this point is then marked by pressing the rubber bulb with the left hand. My practice is to start the tracing on the left side by dotting out the upper outer limit of the aorta, then continuing downwards and marking the other curves of the heart until the entire left side is outlined. To expose the lowest part of the apex, the patient is asked to hold his breath at the end of deep inspiration. The upper right border of the aorta, then the remainder of the right border of the heart, are mapped out down to the diaphragm. Finally, the line of the diaphragm is mapped out on each side. The cardiac contour is best dotted out during the time of systolic contraction. There is no danger of X-ray burns since the amount of current used is small (about 10 amperes). The entire orthodiascopic tracing generally requires about two minutes. During the examination and for a minute or two thereafter, the observer can carefully study the heart action, and thus not only gain important data regarding the various types of contractility, especially of the left ventricle and aorta, but also fluoroscopically study shadows, the interpretation of which may be uncertain in a roentgenogram.

The details of the marking mechanism are shown in Fig. 203 (Plate XVIII).

Orthodiascopic Tracing of the Normal Heart.—The contour of the normal heart and aorta is an irregular oblique ovoid; its larger end is directed downwards and to the left (Fig. 204). The outline is made up of several curves formed by different portions of the heart. On the **right** side above, we find the ascending aorta, which forms a curve with a slight outward convexity (*A 1*, Fig. 204). Above it, the great vessels are sometimes seen as indefinite shadows. Very rarely, the superior vena cava is visible as an attenuated shadow stretching across the aorta. Below the ascending aorta is the curve formed by the right auricle (*R.A.*, Fig. 204). It is usually a well-defined arc and forms an acute angle with the arch of the diaphragm. In cases of extreme right ventricular enlargement, or in pericarditis with effusion, this angle may be obliterated. The **left-sided silhouette** is composed of four more or less distinct curves. From above downwards, these are the aortic arch (*A 2*, Fig. 204), the pulmonary artery (*P.A.*), the left auricular appendix and auricle (*L.A.*), and the left ventricle (*L.V.*). The arch of the aorta protrudes slightly beyond the left sternal border, describing a convex arc; if the aorta curves sharply downwards and backwards to reach the spine, this arc assumes a knoblike prominence. The pulsatile excursions are slight (about .5 centimeter) and require careful scrutiny in order to identify them. The average length of the arch, visible as a separate shadow, is from 3 to 5 centimeters. The left margin of the descending aorta can sometimes be traced down-

wards for several centimeters as a lighter shadow; in exceptional instances, it is dimly seen as a pulsating shadow behind the body of the left ventricle. Beneath the aortal arch and distinguished from it as a much smaller and less convex silhouette is the pulmonary artery; its average visible length is from 2 to 3 centimeters. The contour of the pulmonary artery is distinguished from that of the underlying left

auricle by the difference in pulsation time. The left auricular curve is obtusely oblique and variable in size; it consists mainly of the left auricular appendix, but sometimes also includes part of the left auricle. The left ventricular curve constitutes the greater portion of the left ventricle, and forms its ovoid end. The extent to which the left ventricle is visible depends upon the shape of the heart and the mobility of the diaphragm. In stout individuals with thick abdominal walls, diaphragmatic excursion is usually limited, and the apex of the heart may not be visible; in thin individuals with good diaphragmatic mobility the entire left ventricular surface including the apex is plainly seen at the end of inspiration.

Orthodiascopic Standards. —

In order accurately to determine the standard size of the heart, numerous statistical studies and measurements of orthodiascopic

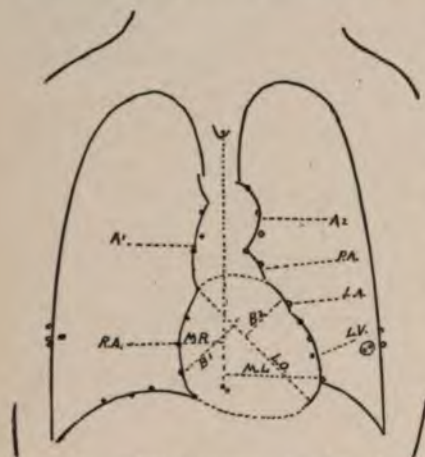


FIG. 204. — Orthodiascopic tracing of the normal heart.

- A 1 = ascending aorta;
- R.A. = right auricle;
- A 2 = aortal arch;
- P.A. = pulmonary artery;
- L.A. = left auricular appendix;
- L.V. = left ventricle;
- M.R. = greatest distance from median line to the right;
- M.L. = greatest distance from median line to the left;
- L.O. = longest oblique diameter of the cardiac ovoid;
- B 1 + B 2 = greatest width of the orthodiascopic tracing.

tracings have been made. For this purpose, oblique and other diameters of the cardiac ovoid (Fig. 204) have been devised and measured; from these, attempts have been made to establish normal cardiac areas. The diameter representing the greatest width of the heart ($B 1 + B 2$, Fig. 204) depends for its accuracy upon the left auricular curve; this is a variable quantity even in normal individuals and hence unreliable as a standard for measurement. Some observers have, therefore, chosen the largest oblique diameter ($L.O$, Fig. 204) and the distance of the cardiac borders from the median line ($M.R$, $M.L$) as being more exact. These axes have been studied principally by Groedel, Dietlin, and Veith in individuals of both sexes, of various ages, weights, and size, with varying types of thorax, and following different voca-

tions. Groedel has found the following figures in the vertical orthodiagrams :

	M.R	M.L	L.O
In the adult male	4.6 cm.	8.4 cm.	14 cm.
Male youths	4.1 cm.	7.8 cm.	12.7 cm.
Female adults	3.9 cm.	8.0 cm.	12.9 cm.
Young females	3.7 cm.	7.2 cm.	12.1 cm.

In these measurements, a certain correlation has been found between the cardiac area, and size and weight of the individual. However, there are important considerations which tend to lessen the value of measurements as standards. Size, weight, type of thorax, and occupation are factors which have already been mentioned. In these alone there is a wide range of maximal and minimal measurements which

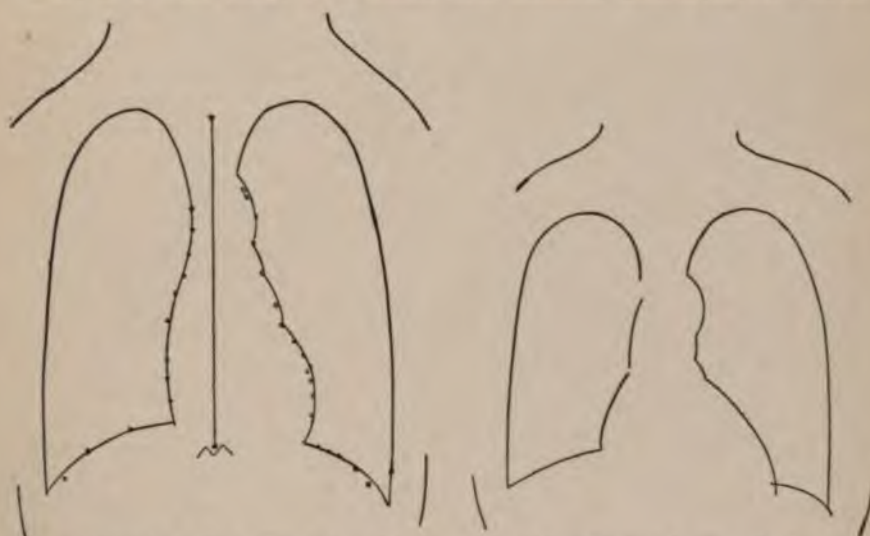


FIG. 205.—Orthodiascopic tracing of long and slender heart.

FIG. 206.—Copy of an orthodiascopic tracing of a broad heart.

may amount to more than 3 cm. Another factor capable of producing wide variations from the standard and still be within the normal is that of different types of cardiac contour which do not correspond with the muscular make-up of the individual. The extremes are those with long, slender, graceful hearts scarcely resting upon the diaphragm, and those whose hearts are broad, lying flat along almost their entire lengths. For example: in a youth of 19, tall and slender (Fig. 205) $M.R=3$ cm.; $M.L=6$ cm.; the other is the orthodiagram of a robust male of 35 (Fig. 206). Both are healthy and have normal hearts. Marked dif-

Recesses in all diameters are immediately apparent by reference to the diagrams. An added consideration, already alluded to, tending to lessen the value of measurements as comparative standards, is the difference produced by movements of the diaphragm. When excursion of the latter is limited, less of the heart is uncovered, its ovoid contour appears flattened, and the transverse measurement is correspondingly enlarged. With a mobile diaphragm the reverse is usually true. From all these facts it is apparently impossible to establish a normal mathematical orthodiascopic standard of measurement. This statement,

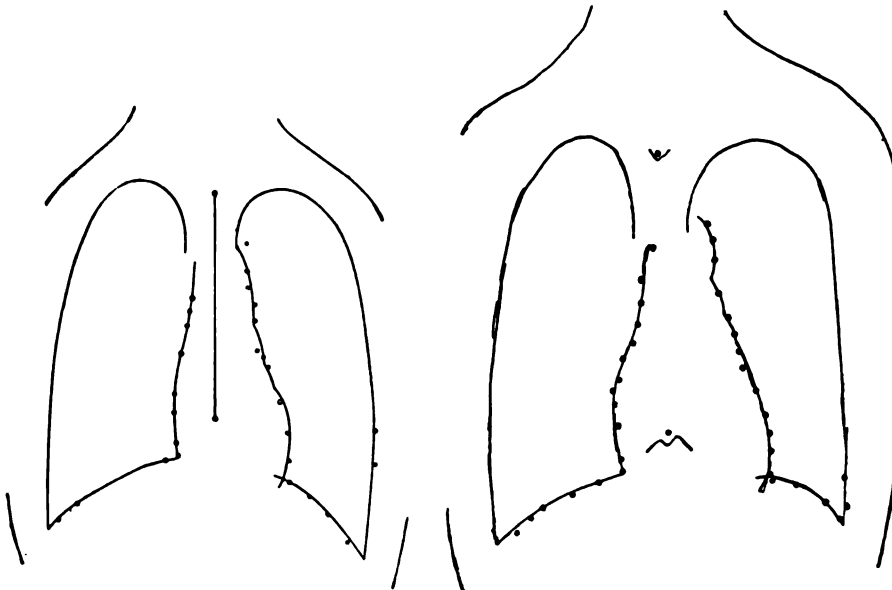


FIG. 207.

FIG. 208.

FIGS. 207, 208. — Orthodiascopic tracings of abnormally slender hearts resting slightly on the diaphragm (the 'drop' heart).

however, does not preclude such general conclusions as that 'normal' hearts may be unusually large or small, or that pathologically enlarged hearts often overstep the somewhat ill-defined normal limits. There is naturally no doubt of the value of orthodiascopy in the determination of marked variations in the size of the heart.

The Narrow and the Broad Heart. — Returning to a discussion of the two types of cardiac silhouette (Figs. 205, 206) — the abnormally narrow and the abnormally broad — there is not only a difference in size but also a marked difference in form. The left ventricle of the broad heart is flat, lies close to the diaphragm, and forms a flattened ovoid. The other type is ellipsoid, and rests lightly upon the diaphragm; Figs. 207 and 208 are additional examples. One is that of a man of 25 suffering from bronchial asthma (Fig. 207); the other that of a woman of

26 suffering from marked vasomotor symptoms (Fig. 208). This type, sometimes called the "drop heart," has assumed considerable clinical importance, for it has been found in patients with visceroptosis, is regarded as one of the characteristics of "habitus asthenicus," and is apparently the type of cardiac hypoplasia described in status thymolymphaticus. The symptoms I have found in these individuals are those connected with vasomotor instability — flushes, pale and cold extremities, dizziness, etc. These are often erroneously interpreted as due to a 'weak heart' (Chapter XX).

Orthodiascopy in Mitral Disease. — Orthodiascopy has been used as a method of establishing the diagnosis of valvular lesions upon the assumption that these produce typical changes in cardiac contour. With reference to mitral disease, it is probably true that many advanced decompensated patients

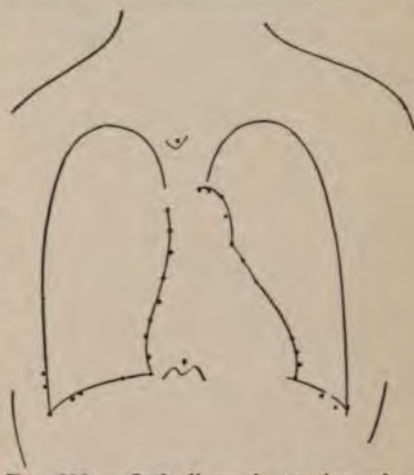


FIG. 209. — Orthodiascopic tracing of a woman of 30 with a compensated mitral regurgitant lesion showing normal cardiac contour and size.



FIG. 210. — Orthodiascopic tracing of a girl of 17 with a compensated double mitral lesion. The tracing is normal in size and contour.

conform in a general way to an orthodiascopic type, but those with quiescent and compensated lesions may possess hearts of normal size and contour. Two illustrations are given in Figs. 209 and 210. The former was taken from a woman of 30 with a mitral regurgitant lesion of ten years' duration; the latter, from a girl of 17 with a double mitral lesion of four years' duration. In both, there were typical physical signs of the respective valvular lesions; both were compensated, the patient with the double mitral was still suffering from rheumatic manifestations at the time the orthodiascopic tracing was taken. Decompensated double mitral lesions with auricular fibrillation are those which usually conform to a general orthodiascopic type. The aorta may retain its normal outline or be somewhat dilated, even in the absence of physical signs of an aortic lesion. The remainder

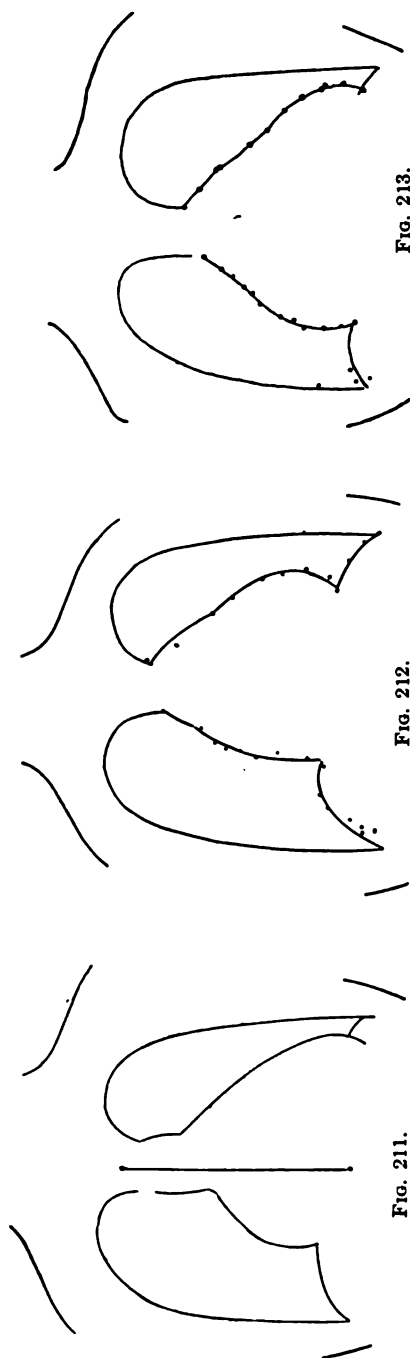


FIG. 211.

FIG. 212.

FIG. 213.

FIGS. 211, 212, 213. — Examples of globular hearts of varying degrees of rotundity and size. From three cases of decompensated double mitral lesions with auricular fibrillation. In Fig. 211 the aortic curve is normal.



FIG. 214. — Globular heart of extreme size. Copy of an orthodiastolic tracing of a boy of 11 suffering from a double mitral lesion and auricular fibrillation.

FIG. 215.

FIG. 216.

FIGS. 215, 216. — Orthodiastolic tracings showing a somewhat globular heart with moderate enlargement. From cases with double mitral lesions and auricular fibrillation.

of the cardiac contour forms one large fused outline — the globular heart ('Kugelherz'), consisting of the dilated pulmonary artery, left auricular and ventricular curves on the one side, and a ballooning out of the right auricular curve on the other.

Figures 211, 212, 213 are examples showing varying degrees of size and rotundity of outline from three cases of decompensated double mitral lesions with auricular fibrillation. All the patients were adults. In one (Fig. 211), the aortic curve is normal; in the others, it is dilated. Another interesting example of the globular heart (Fig. 214) is the orthodiagram of a boy of 11 with a decompensated double mitral lesion and auricular fibrillation. Occasionally, the orthodiascopic tracings of patients with these lesions show neither extreme enlargements nor a definite

rounded outline, thus: Figure 215 is the tracing of a man of 50 who only recently developed auricular fibrillation; Fig. 216 is from a woman of 46 who has had the valvular lesion and arrhythmia for many years.

Other variations from the

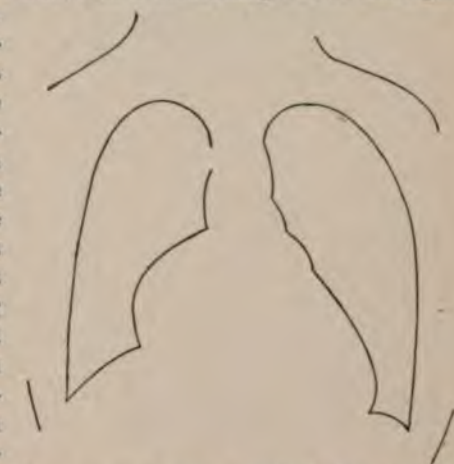


FIG. 217. — Extreme rounded right-sided enlargement. The left contour is enlarged downward. From a case of double mitral lesion, auricular fibrillation, and cardiac failure.

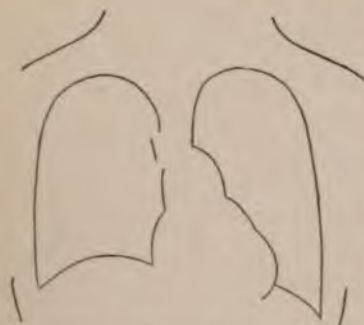


FIG. 218. — Copy of orthodiascopic tracing of a woman of 55 with a double mitral lesion of 20 years' duration. There is only very slight left-sided enlargement.

typical globular form are sometimes found in chronic cases of double mitral disease with fibrillation in which there is disproportionate right- and left-sided enlargement. For example, the tracing in Fig. 217 is from a patient of 35 with an old double mitral lesion, frequently decompensated, and with auricular fibrillation. There is a sharply rounded right-sided enlargement, the left side is enlarged downwards and outwards; the resultant silhouette is an irregular ovoid.

Of great interest from the orthodiascopic standpoint are those patients with chronic double mitral lesions without auricular fibrillation in whom

there has never been any severe break of compensation. The tracings are sometimes normal in configuration and size, as in a case already described (Fig. 210), or the enlargement is too slight to be of

diagnostic significance. Thus, a woman of 45 has had a double mitral lesion over twenty years. Recently she developed occasional extrasystoles accompanied by a subjective feeling of "weakness" in the chest. Otherwise, she has never had any cardiac complaint.

The orthodiascopic tracing (Fig. 218) shows only very slight left-sided enlargement.

Regarding the orthodiagrams in patients with mitral regurgitant lesions, there may be no abnormal change of cardiac outline, particularly when the lesions are recent and quiescent, or are fully compensated. One example has been mentioned above (Fig. 209).

Another instance is the tracing of a tall, well-built lad of 18 (Fig. 219), who recently recovered from the acute manifestations of mild rheumatic mitral regurgitation.

In chronic mitral regurgitation with decompensation, the orthodiascopic tracing is usually ovoid or somewhat circular in shape. The longest axis lies diagonally, the larger end corresponding to the left ventricle. The elements constituting the abnormal form are moderate enlargement of the left auricular, ventricular, and the right auricular curves. An instance of this is seen in Fig. 220, a female aged 35 with a rheumatic mitral regurgitant lesion; the orthodiagram shows an ovoid heart with moderate dilatation of the left border. If decompensation is extreme, the entire contour becomes circular.

Orthodiascopy in Rheumatic Aortic Disease.— With reference to rheumatic lesions of the aorta, one of the chief fluoroscopic characteristics is the degree of the aortic excursion, the aortic fling. This is observed in patients with double aortic lesions whether compensated or not. As a rule, the aorta itself is not permanently enlarged, although

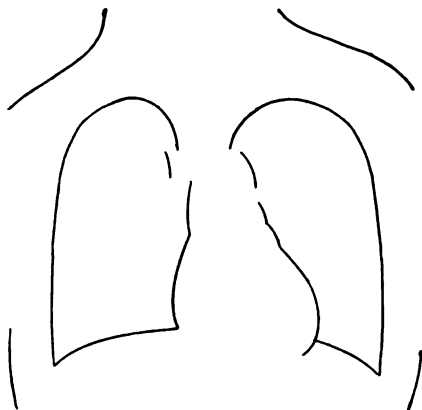


FIG. 219. — Normal orthodiascopic tracing of a tall boy of 18 suffering from a recent mitral regurgitant lesion.

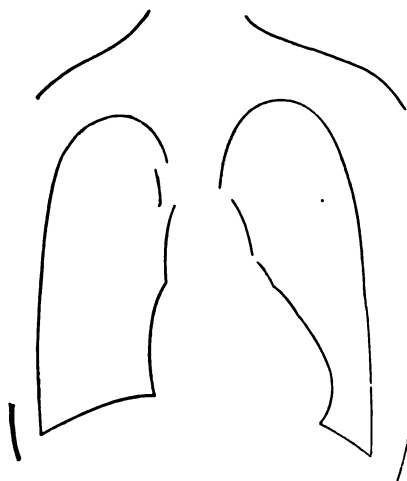


FIG. 220. — Orthodiagram of a woman of 35 with a mitral regurgitant lesion showing a somewhat ovoid contour with moderate dilatation to the left.

this impression is gained from the occasional violent systolic expansion. There appears to be some relation between the clinical severity of the disease as gauged by the dyspnoea and cardiac reserve power, and the degree of aortic fling. In cases severely ill, the entire aorta, the ascending, arch, and descending portions pulsate so violently as to resemble an aneurism; those less ill usually show excursion limited to individual sections of this vessel. Another fluoroscopic characteristic of aortic lesions is the sharp, vigorous left ventricular contraction, with a distinct lifting motion of the apical region. When left ventricular hypertrophy is extreme, the contraction of the apical region is so marked as to appear separate from the remainder of the left ventricle. In general, it may be stated that the ventricular excursion during systole is greater in aortic valvular lesions than in the other types of cardiac disease; this is sometimes of diagnostic significance in suspected aortic disease with indistinct and atypical clinical signs.

The change in cardiac contour found in aortic valvular lesions is

more constant than in mitral disease. The apical region is enlarged and obtuse, and the apex itself is often found near the axillary line. The body of the left ventricle is egg-shaped. The right auricular curve is moderately increased in convexity, the entire auriculo-ventricular outline presents an abnormally broadened, flat, somewhat egg-shaped oval, with the wide end at the apex. In conjunction with the curves formed by the aorta and pulmonary artery, the entire cardiac outline roughly resembles the "duck-shaped heart" described by Groedel.

Several typical illustrations of double aortal lesions follow:

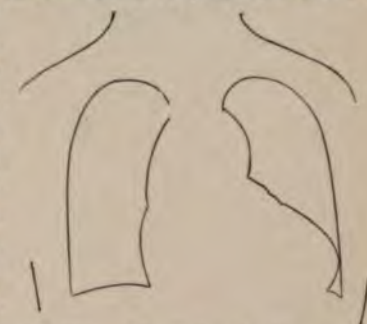


FIG. 221. — Typical orthodiagram of old rheumatic double aortic lesion. From a woman of 45 showing extreme left ventricular enlargement and dilatation of the first part and arch of the aorta.

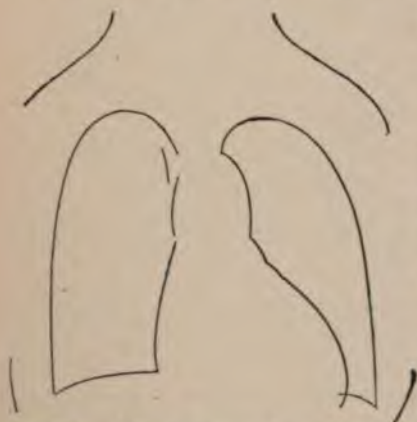


FIG. 222. — Orthodiagram of a patient with aortic regurgitation. From a boy of 19, showing left ventricular hypertrophy and moderate enlargement of the aortic arch. The pulmonary and left auricular curves are small.

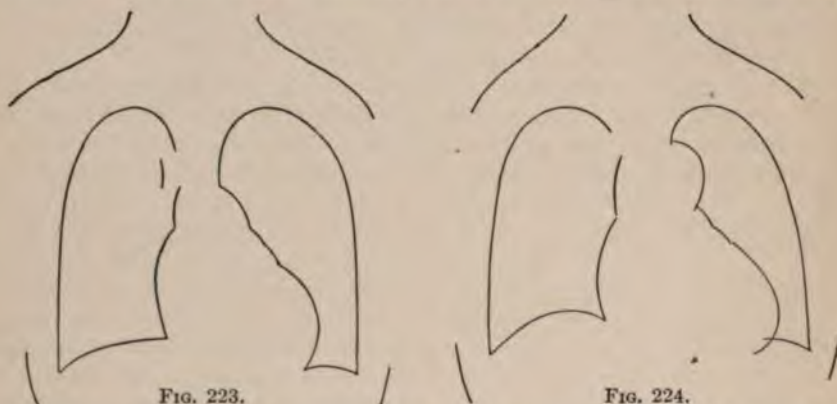
Figure 221 is the orthodiagram of a woman of 55 with an old rheumatic history. There was dilatation of the first part and arch of the aorta. The entire left ventricle was greatly hypertrophied, its outlines enlarged, the pulmonary curve flat-

tened, the left auricular curve very small. Fluoroscopically, there was very marked aortal fling of the entire visible aorta.

Figure 222 is the tracing of a boy of 19 with a rheumatic aortic lesion of twelve years' duration. There was marked left ventricular hypertrophy, the pulmonary and left auricular curves were small, there was moderate dilatation of the aortic arch; fairly marked aortal fling was present. Whether the right-sided enlargement was due to the right auricle itself, or whether an enlarged left ventricle pushed the right auricle abnormally to the right, was not clear.

While the entire foregoing description applies especially to double aortic lesions, with some differences it also applies to aortic stenosis alone. In the latter the size and shape are similar to the former, but enlargement of the various curves is not so extreme, and the aortic fling is not as marked.

Orthodiascopy in Combined Aortic and Mitral Disease.—In combined aortic and mitral lesions, the orthodiascopic tracing usually



FIGS. 223, 224. — Orthodiagrams of two cases of double mitral and aortic lesions. The outlines are characteristic of the latter in Fig. 224. Fig. 223 is not characteristic of either lesion.

follows the type of the one clinically predominant. Thus, Figs. 223 and 224 are taken from two cases of mitral regurgitation and double aortic lesion. The first is that of a girl of 12 in whom endocarditis developed at the age of 5; the second, that of a girl of 19, the duration of whose endocarditis is not known. The first is not characteristic of either lesion; the second shows the characteristics of aortic lesions from which this patient chiefly suffered.

ORTHODIASCOPY IN AORTITIS

Aortitis with secondary involvement of the aortic cusps belongs in a different category from the rheumatic valvular lesions. Although

calcification and thickening of the aorta do not necessarily imply enlargement and dilatation, the latter is the rule in aortitis. This lesion may be confined to the first part (the ascending aorta), to the arch, or to the descending thoracic aorta; or may involve the entire thoracic and even part of the abdominal aorta. Hence, it is important to distinguish aneurismal dilatations and enlargements of various parts of this vessel.

Moderate dilatation and enlargement of the first or ascending part of the aorta are characterized by an abnormally broad shadow which originates below the normal level. The left border of the arch is found beyond its usual limits and, in

addition, may be sharply curved. The entire aortal shadow thus becomes abnormally broad. This type of enlargement accompanies aortitis from any cause — senile arteriosclerosis, chronic nephritis with hypertension, luetic aortitis; it is rarely found in rheumatic aortic valvular disease.

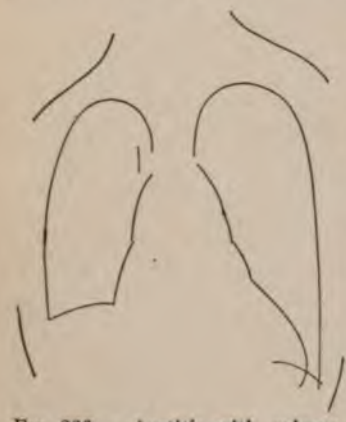


FIG. 226. — Aortitis with enlargement of the first portion and arch of the aorta, and left ventricular dilatation. From a patient of 65 with symptoms of general arterial sclerosis appearing one year after a very severe grippe infection.

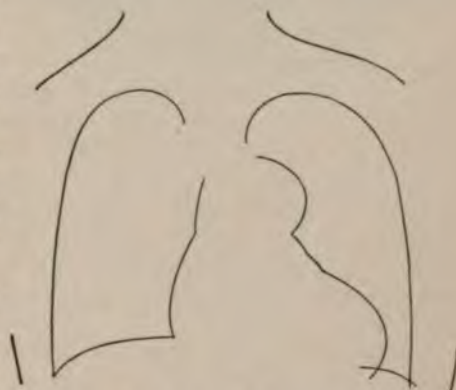


FIG. 225. — Dilatation of the aortal arch; left ventricular hypertrophy. From a male patient of 45 with chronic nephritis and moderate hypertension.

As with standard of measurements for the cardiac orthodiagram, similarly there are several objections to a standard measurement for the normal aorta. Some of these are the indefinite point of origin of the aorta, the difficulty at times of delimiting its borders, the high or low position of the arch, the size and shape of the heart of each individual. In aortic dilatation of moderate degree, the diagnosis may therefore depend upon **abnormal contour** rather than upon abnormal size of the vessel. In moderate dilatation of the ascending aorta there is a slight outward ballooning of the right aortal outline; in that of the arch, the curve is broadened; in that of the descending

part of the arch, there is distinct widening of the entire aorta. Finally, there are dilatations confined to the descending thoracic aorta; the latter then assumes a spindle-like or diffuse enlargement, which lies

mainly behind the ventricles. The various types of aortic enlargement are sometimes best seen and recognized by turning the patient in different directions in order to obtain lateral and diagonal views of the aorta.

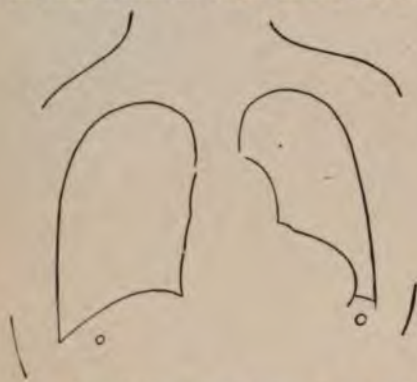


FIG. 227. — Dilatation of the aortic arch and left ventricular hypertrophy. From a patient of 60 with arteriosclerosis.

This is of particular value in differentiating aortal shadows from other structures; for example, from tumors, which in a sagittal direction simulate the aorta in area and position.

Orthodiagrams of various types and degrees of aortitis with accompanying cardiac hypertrophy and of aneurismal dilatations are shown in Figs. 225-232, Fig. 233 (Plate XIX), Fig. 234 (Plate XX), Fig. 235, with a brief summary of their etiology and clinical diagnosis.

CONGENITAL CARDIAC MALFORMATIONS

Aortic stenosis is one of the more frequent congenital malformations. It usually occurs about $2\frac{1}{2}$ cm. above the implantation of the aortal cusps and constricts the vessel to a varying degree. It may be the only congenital cardiac anomaly. As in other congenital defects of the heart, the patients during childhood or adolescence often develop endocarditis of the aortic or other valves, conditions which partially or entirely mask the stenosis.

The following are brief reports of cases with orthodiagrams of the foregoing congenital defect:

A male, age 21 years, had had a very severe attack of scarlet fever when 10 years old; it was followed by endocarditis affecting the mitral and aortal valves. The patient was then abed for several months. Except for occasional attacks of hematuria, he felt quite well until his twenty-first

year. At that time he complained of lassitude for several weeks, during which there were no rheumatic manifestations or fever. Upon examination, the heart was found enlarged to the left; there was an area of dullness over the second and third right interspaces where a

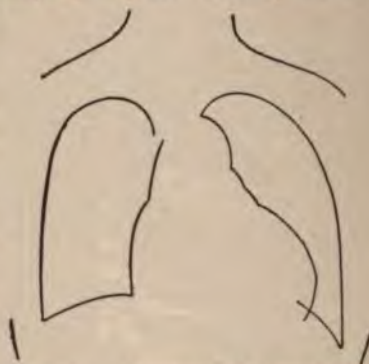


FIG. 228. — Aortitis of the first portion and arch of the aorta, and extreme left ventricular hypertrophy. From a man of 50 with cardiosclerosis and decompensation. The first clinical symptoms occurred six months after a very severe streptococcus pneumonia.

PLATE XIX



FIG. 233. — Photograph from X-ray plate, showing aneurismal dilation of the descending thoracic aorta.

PLATE XX

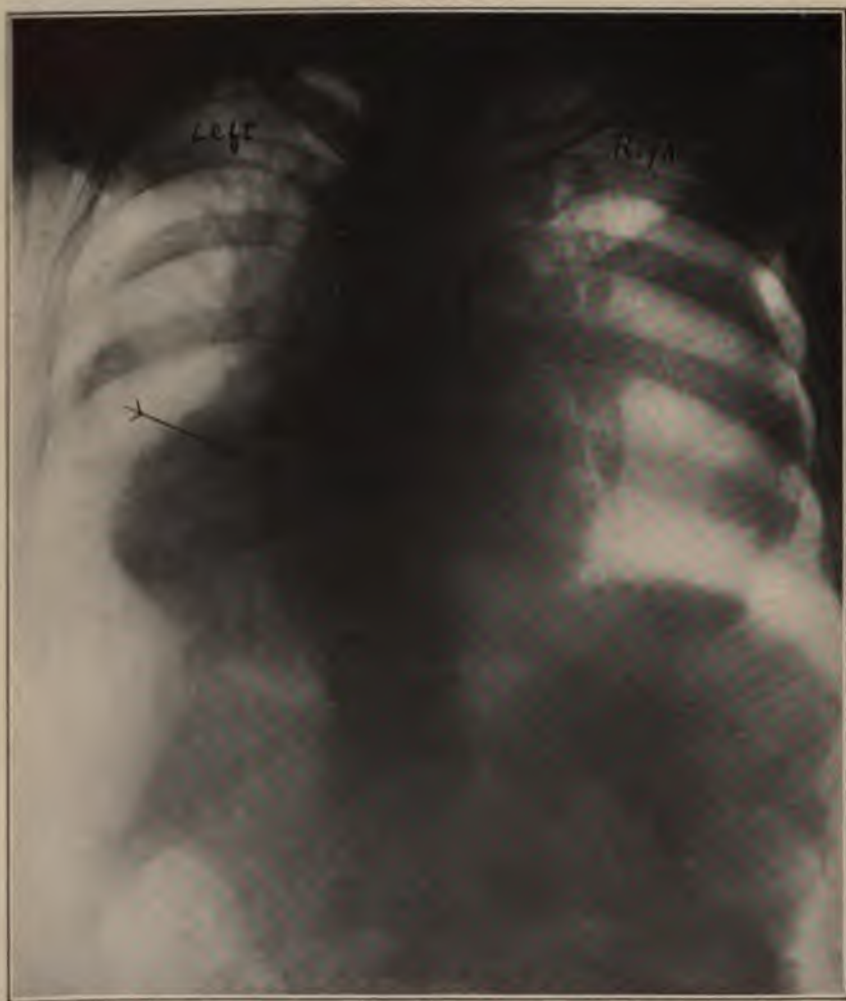


FIG. 234. — Photograph from Roentgenogram of aneurismal dilatation of the descending thoracic aorta; marked left ventricular hypertrophy. From a woman of 50 with a positive Wassermann reaction of the blood.

loud systolic and a fainter diastolic murmur were heard. There was also the auscultatory evidence of a double mitral lesion. The Wassermann blood reaction was negative. Fluoroscopic examination showed aneurism of the first portion of the aorta (Fig. 235). The diagnosis of

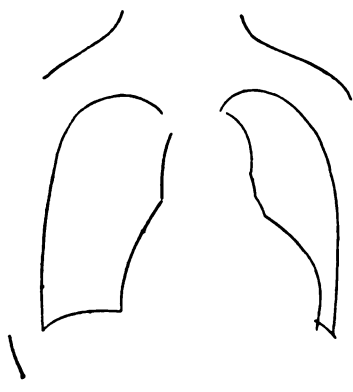


FIG. 229. — Luetic aortitis affecting the arch. Left ventricular hypertrophy. From a patient of 60 with a luetic history and a positive Wassermann blood reaction.

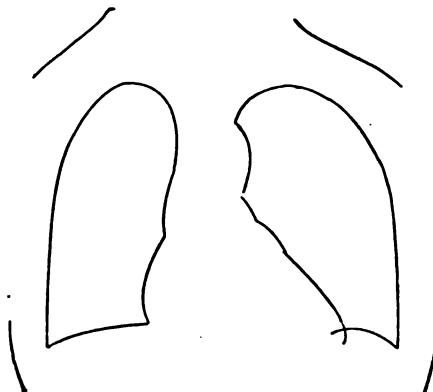


FIG. 230. — Enlargement and low implantation of the first portion of the aorta; moderate enlargement of the left ventricle. From a male patient of 58 with chronic nephritis and myocarditis.

congenital aortic stenosis was made. Some weeks later, the patient developed acute gastric symptoms with fever. A tentative diagnosis of streptococcus viridans infection was made; this was confirmed two days later by positive blood culture. Despite immediate treatment with autogenous vaccines and sensitized horse serum, the patient died within a few months. At necropsy, the mitral and aortic cusps were found encrusted with granulomatous masses, some of which were imbedded in the sinuses of Valsalva. There was moderate congenital constriction of the aorta 3 cm. above the cusps. There were no other congenital malformations. It is of great interest to note that though serum therapy was begun within a very few days after the onset of the acute symptoms and was systematically continued, there was absolutely no evidence at necropsy that the progress of the disease had in any wise been checked.

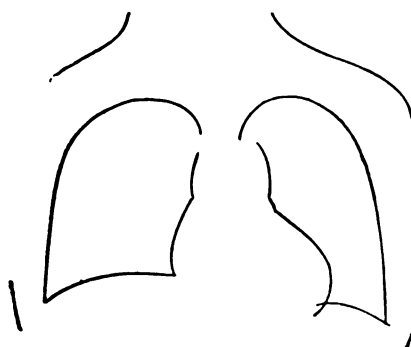


FIG. 231. — Luetic aortitis affecting the arch; left ventricular hypertrophy.

A woman, aged 51, had scarlet fever when 6 and "heart disease"

when 7 years old. From that time until the present, she has had frequent attacks of decompensation. From her account, she appears to have had attacks of paroxysmal tachycardia; otherwise, the pulse

had been regular until quite recently. She then developed rapid and irregular heart action, orthopnea and anasarca. The electrocardiogram revealed auricular fibrillation; there was definite auscultatory evidence of a double mitral lesion. At the first examination, the sounds at the right base appeared normal. With the restoration of compensation and decrease of the ventricular rate, a soft systolic and a fainter diastolic murmur were heard over the right base; there were no additional signs of an aortic lesion. Fluoroscopic examination showed a large pulsatile enlargement of the first



FIG. 233. — Diffuse aneurismal dilatation of the descending thoracic aorta. From a patient of 60 with a history of a luetic infection 40 years ago, and a positive Wassermann reaction of the blood.

portion of the aorta (Fig. 237). From the history and fluoroscopic findings, it seems most probable that the aortic enlargement was due to congenital aortic stenosis.

PATENT DUCTUS ARTERIOSUS OR DUCTUS BOTALLI

This congenital defect is not rare. Cardiac symptoms are not always present; when they are, the usual history is that the patients

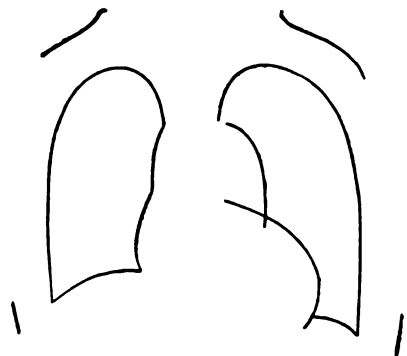


FIG. 235. — Aneurismal dilatation of the arch and descending thoracic aorta.

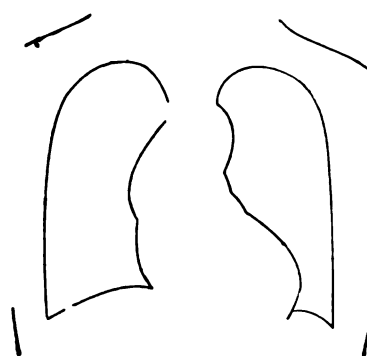


FIG. 236. — Aneurism of the first portion of the aorta due to congenital aortic stenosis.

were born cyanotic. The cyanosis is increased by crying, by exposure to cold, and, in older children, by exertion and exercise. Upon percussion, a slight extension of cardiac dullness in the second left inter-

space (Gerhardt's area of dullness) can sometimes be mapped out. A rough systolic thrill is felt, and a loud, rough systolic murmur is heard, most intensely over this area. The murmur is usually transmitted over the entire precordium and into the carotids. In addition, there is sometimes a loud diastolic murmur most prominent in the left upper interspaces near the sternum; the double murmur thus produced is called a "machinery murmur." The presence and intensity of the diastolic murmur apparently depend upon the amount of blood which regurgitates through the patent duct, and, in its mechanism, resembles aortic insufficiency; indeed, capillary pulsation has been described as an occasional accompaniment of this lesion. The orthodiascopic picture in typical cases of patent ductus arteriosus is that of a vigorously pulsating

aortic arch increased in size, beneath which is found an enlarged, flattened, or protuberant overacting pulmonary artery. The duct itself is rarely visible because of its small size and frequent involvement in pericardial adhesions. The latter was found in two autopsy specimens I have seen.

The following is an illustrative history, with clinical and orthodiascopic findings, of a patient with patent ductus Botalli. Male, age 16, tall and spare, was born a "blue baby." He had scarlet fever when seven years old. He had never been able to play actively or to run. He frequently complains of palpitation; he has a productive cough and has had hemoptyses. His face and extremities are cyanotic, his lips almost black, his fingers markedly clubbed. There are vigorous carotid and jugular pulsations and a strong visible ventricular

impulse; the apex is most prominent in the fifth interspace. A strong systolic pulsation is seen in the second and third left interspaces just outside the sternal border. There is a marked palpable carotid thrill felt

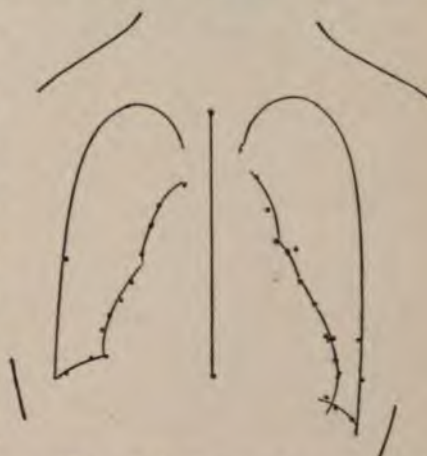


FIG. 237. — Orthodiascopic picture of a patient with a double mitral lesion and aortic aneurism, the latter probably due to congenital aortic stenosis.

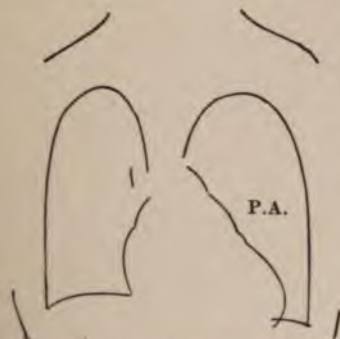


FIG. 238. — Orthodiascopic picture of a boy of 16 with patent ductus arteriosus. The curve of the pulmonary artery (P.A.) is extremely enlarged and flattened.

almost up to the angle of the jaw. There is also a rough thrill most prominent in the second, third, and fourth left interspaces, less marked in the second and third right interspaces. A definite "Gerhardt's area of dullness" can be mapped out by percussion. Upon auscultation, a loud systolic murmur is heard over the entire precordium, and over the chest posteriorly. The murmur is especially loud and rasping over the second and third left interspaces and over the manubrium sterni. The fluoroscope reveals a vigorously overacting aorta and pulmonary artery; the latter is extremely enlarged and flattened (Fig. 238). The electrocardiogram shows a negative *R* deviation in the first and second leads, an evidence of right ventricular preponderance.

PATENT INTERVENTRICULAR SEPTUM

This congenital anomaly is extremely uncommon and very rarely exists as an isolated defect. Cyanosis is the most prominent clinical sign; its degree depends upon the size of the interventricular aperture, and upon the presence of other congenital defects — usually pulmonary atresia. The extremities are clubbed, there is a marked coarse systolic thrill to be felt, and a corresponding rough murmur to be heard, over the entire precordium; the maximum intensity of the murmur and thrill is over the xiphoid and epigastrium. There is also vigorous epigastric pulsation. In the orthodiagram the left auricle and ventricle are hugely dilated and rounded, the heart is also enlarged to the right; because of atresia of the pulmonary artery its pulsation may be slight or absent.

A unique instance of patent interventricular septum in a child with congenital dextrocardia came under my observation. The history follows:

A male, age $3\frac{1}{2}$ years, was the youngest of nine children; he had measles and pertussis when two years old. The mother said the child had always been "blue" and "short of breath," but never sufficiently dyspnoeic to be bedridden. The child appears well nourished. The extremities, lips, face, and conjunctivæ are cyanosed; the conjunctival blood vessels are congested; there is distention of the superficial veins of the chest and abdomen. The fingers and toes are clubbed. There is a vigorous, heaving systolic impulse in the right axillary line and in the epigastrium. A strong, marked systolic thrill can be felt over the entire precordium; it is roughest and most prominent at the lower end of the sternum and in the epigastrium. Over the former there is a loud, rough systolic murmur becoming fainter over the remainder of the chest, anteriorly and posteriorly. It is also transmitted along the carotids. There is no Gerhardt's area of dullness in the second and third right interspaces. The liver and spleen are not transposed; the gastro-intestinal canal, as shown by bismuth roentgenograms, is in its normal position. Fluoroscopy of the chest

reveals an enlarged and vigorously pulsating aorta. Beneath it, the usual pulsating curve, representing the pulmonary artery, is absent. In its stead, there is a tremendously dilated, prominent, and knob-like 'left'¹ auricle. The 'left' ventricular shadow is also very much enlarged; its area cannot be definitely separated from the superimposed auricle, although their non-synchronous pulsations can be readily determined. The 'right' auricle is enlarged; beneath it, the beginning of the 'right ventricular' shadow is seen deeply depressing the diaphragm during inspiration. From this finding, it is probable that the 'right' ventricle is enlarged. The orthodiagram (Fig. 239) showed a greatly dilated aorta, rounded and prominent, and coalescence of the 'left' auricular and ventricular curves. The 'right auricular' curve is also considerably enlarged. The electrocardiogram shows the typical downward deviation of the *P*, *R*, and *T* waves in the first lead, pathognomonic of congenital dextrocardia. A diagrammatic representation of the probable circulation in this case is also shown in Fig. 240.

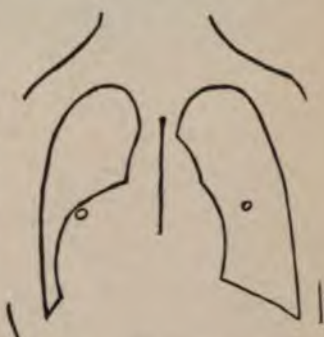


FIG. 239. — Orthodiagram of a child with congenital dextrocardia and patent interventricular septum. It shows an enlarged and broadened aortal curve and enlarged 'right'¹ auricular curve, coalescence of enlarged and knob-like left auricular and ventricular curves, and absence of pulmonary prominence.

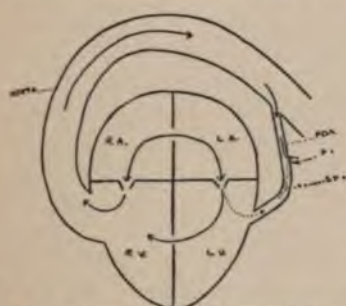


FIG. 240. — Diagram of probable cardiac circulation in the patient DL. Solid arrows = main circulation. Dotted arrows = minor circulation.

R.A. = right auricle;
R.V. = right ventricle;
L.A. = left auricle;
L.V. = left ventricle;
P.D.A. = patent ductus arteriosus;
P.C. = pulmonary circulation;
S.P.A. = stenosed pulmonary artery.

usually applied to the normal heart in its normal position; that is, 'left' will refer to the larger, and 'right' to the smaller ventricle with its corresponding auricle.

PATENT FORAMEN OVALE

When present as an isolated anomaly, and of only moderate size, this interauricular septal defect rarely produces clinical signs or diagnostic symptoms. As proven at necropsy, patients with even large defects have presented during life no signs, clinical or otherwise, upon which a correct diagnosis might have been based. Cyanosis is rarely present; occasionally, systolic or diastolic murmurs are heard over the third left interspace. Orthodiascopic tracings present nothing characteristic.

¹ To avoid confusion in the description of this case of congenital dextrocardia, 'left' and 'right' are used in the sense as

Of congenital lesions in general, it may be broadly stated that patients with a single congenital defect are those who are most apt to survive and reach adult life. If more than one congenital anomaly is present, the chances for longevity are correspondingly diminished.

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CHAPTER IX

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CHAPTER X

PHYSICAL EXAMINATION OF THE HEART

EACH of the usual methods of clinical examination — inspection, palpation, percussion, and auscultation — yields valuable information in appropriate cases. Ordinarily, most emphasis is placed upon auscultation and percussion, less upon inspection and palpation. In my opinion the latter, though frequently neglected as routine methods of examination, are often of diagnostic and prognostic importance. Least valuable and most deceptive of all methods is information gained by percussion.

INSPECTION

Inspection of the chest and neck frequently reveals important data concerning the cardiovascular system.

Jugular Pulsation. — The manner of filling, and the degree of distention, of the jugular veins is best observed by placing the patient in the supine position. Even pulsation of the thyroid veins may be visible. In patients with long-continued decompensation it is common to find the jugular veins tortuous, thickened, engorged, and greatly distended; the situation of the valves is particularly prominent. These evidences of chronic venous stasis are especially marked in old mitral stenotic lesions with auricular fibrillation, or in old patients with decompensated cardiosclerosis. Tricuspid regurgitation from relative insufficiency or valvular disease is sometimes accompanied by engorged or hyperactive pulsatile jugulars, although, as already mentioned, the interposition of the right auricle probably prevents such jugular hyperaction in many instances. In cases of **auricular extrasystoles**, the premature jugular pulsation (a' wave) may occasionally be visible. More often, the coincidence of auricular and carotid waves (composite $a' + c'$ wave, Chapter VII) found in **ventricular extrasystoles** is recognized in the jugulars as abnormally large and premature pulsations. A similar jugular wave, not premature in the time of its occurrence, is present in complete heart block when auricles and ventricles beat synchronously ($a + c$ waves; Chapter VII, Heart Block). This observation is of use in the differentiation, by inspection, between complete and incomplete heart block, for in the latter there are no such superimposed waves. In

auricular flutter, the jugular pulsations are usually too faint and indistinct to be counted; however, in one case that I observed the jugular veins literally throbbed, and were so engorged that the number of pulsations could be clearly discerned at a distance of several feet. In **auricular fibrillation**, in addition to the distention already referred to, the jugulars beat irregularly in consonance with the arrhythmic *c-v* waves.

The Carotid Pulse. — The inspection of the carotid pulse is of great importance. By its vigor, overaction, and pulsatile distention, it is often indicative of aortic disease or of hypertension; if, in addition, the carotids visibly and markedly collapse with diastole, they indicate an aortic regurgitant lesion. Vigorously throbbing carotids also accompany rapid and violent heart action from any cause (for example, exophthalmic goiter). But visible carotid hyperaction with a normal pulse rate in the middle aged or the old is usually evidence of aortitis or of hypertension; its absence, however, does not necessarily exclude these conditions. This type of carotid action is also seen, though rarely, in young individuals with normal hearts; in them, it often corresponds to strong systolic ventricular contractions which can be very readily observed by fluoroscopic examination.

Aortic Pulsation. — Allied to carotid overaction in clinical significance is aortic pulsation, recognized by the pulsatile rise of the tissues in the jugulum. This indicates more advanced aortic changes than does carotid overaction alone. Aortic expansion is most noticeable in aortic regurgitation with left ventricular hypertrophy, and in aneurisms and aneurismal dilatation of various parts of the aorta, especially during the stage of decompensation. On the chest wall, capillary pulsation characteristic of aortic regurgitation may occasionally be seen. A band of dilated venules and capillaries over the lower anterior part of the chest sometimes accompanies aortic lesions, particularly aneurisms or aneurismal dilatations. Large aneurisms that touch, adhere to, or erode the chest wall naturally give rise to visible pulsatile expansion; their usual site is the second and third right interspaces and the interclavicular notch, more rarely the left upper interspaces. Aneurismal dilatations of the aorta are rarely adherent and very infrequently cause erosion. If they touch or approach the chest wall, pulsatile expansion, otherwise not noticeable, may be seen by placing the eye on a level with the chest. By this method, not only dilatations of the first part and arch, but even aneurismal dilatation of the descending thoracic aorta (Chapter XIV) may be discerned as a distinct and separate systolic heave above and partly behind the rise of the left ventricle.

Inspection of the Apical Region. — Regarding the apical and precordial region, important information can be gleaned by inspection alone. In thin-chested individuals with decompensated mitral stenosis, it is often possible to discern an overacting pulmonary artery which, upon auscultation, produces an accentuated and clicking second sound.

Many varieties of abnormal ventricular action are also readily recognized by inspection. Thus ventricular activity is visible over almost the entire precordium in massive left ventricular hypertrophy, in the violent action of tachycardia, in the ventricular dilatation of the acute stages of valvular endocarditis, or in some congenital lesions. An overacting ventricular impulse may be confined to a small area at the apex in mitral lesions, especially in young individuals. One often finds heaving, broad ventricular impulses in old, decompensated mitral lesions. Obviously, the thickness of the chest wall, the posture of the patient, and the illumination are considerations which considerably mask or influence these physical data. In thin-chested individuals, or in women with the left breast well raised, it is sometimes possible to distinguish a double systolic impact corresponding to the reduplicated first sound (*q.v.*) heard on auscultation. Some types of arrhythmias are also recognized by inspection at the apex. The regular, slow ventricular activity of heart block, the irregular ventricular action of auricular fibrillation, the apparent intermission of cardiac activity denoting premature contraction — all these are often distinguishable upon inspection of the apical region. The extrasystolic contraction itself is rarely seen because it is usually too weak to cause a visible ventricular impact. The movement of the apical region during the respiratory phases may likewise be discernible in thin or moderately thick-chested individuals who possess a fair amount of diaphragmatic excursion.

Inspection of the Right Lower Interspaces. — Over the right lower chest, aside from aneurism and aneurismal dilatations already mentioned, very little information can be gleaned by inspection. Right auricular and right ventricular enlargement rarely give rise to visible pulsation. I have met with isolated exceptions, however. One case was that of a boy of 17 with a double mitral lesion and a somewhat enlarged liver. At the time of hospital admission he was suffering from an acute rheumatic exacerbation of endocarditis. To the right of the sternum, in the third and fourth interspaces, there was a visible pulsatile area approximately the size of a pigeon's egg. Over this, there was heard a very loud harsh murmur, a rough palpable thrill was present; both were systolic in time. Fluoroscopic examination showed that the pulsation was due to a greatly enlarged right auricle. The patient died a few weeks later. At necropsy, a double mitral lesion, an hypertrophied right and left ventricle, and a hugely dilated, engorged, and thickened right auricle were found. *In situ* in the chest cavity, the longest measurement of the auricle was 17 cm., its broadest 8 cm. (Fig. 241). In another instance of decompensated double mitral lesion in a woman of 26, intensely dyspnoeic, cyanotic, and with a pulsating liver, there was a similar though less prominent visible auricular pulsation in the third and fourth right interspaces; a thrill was also heard over the same area.

Visible epigastric pulsation in patients with dilated and hypertrophied hearts is fairly common. It is usually attributed to hyper-

trophic enlargement of the right ventricle, but necropsy reports have not always substantiated this supposition (Chapter XV).

Liver pulsation is not infrequent in decompensated mitral lesions, or in relative tricuspid regurgitation. It is less common in aortic lesions. The pulsations may be sufficiently gross to be seen at a distance; at other times, close examination, especially with the observer's eye placed on a level with the patient's abdomen, is necessary in order to discern the pulsating liver. Auricular fibrillation may occasionally be

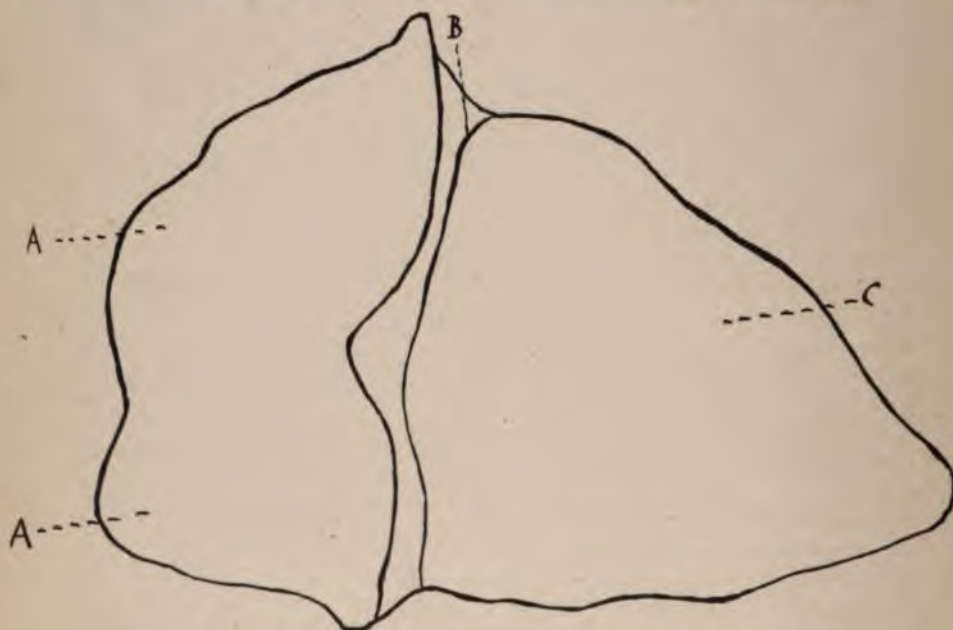


FIG. 241. — Outline of the heart *in situ* (reduced $\times \frac{1}{2}$).
A = dilated right auricle; B = midline of the body; C = enlarged right ventricle.

diagnosed by inspection of the liver, if its pulsations are manifestly and grossly arrhythmic.

PALPATION

Palpation in the neck often substantiates the information gained by inspection. **Extrasystoles** may be felt in the carotids when they are missed at the wrist. Overacting hearts (for example, exophthalmic goiter) often produce palpable systolic carotid thrills; these may be due to whorls and eddies from the violence with which the blood is thrown into the arterial circulation. Such short, sharp thrills must be distinguished from the steadier and longer continued *s accompanying rheumatic aortic stenosis and arteriosclerotic

roughening and thickening of the aortic valves and walls. The typical quick filling and collapsing pulse of aortic regurgitation can also be readily diagnosed by palpation alone. A point insufficiently emphasized is the knowledge regarding arteriosclerosis which may be gained by carotid palpation; the condition of the external carotids, whether tortuous, thick, hard and non-elastic, or the reverse, is thus often determined. Similar information is occasionally derived by palpation of the common carotid.

Palpation of the Aorta. — With a normal cardiovascular system and an aortal arch situated moderately high in the chest, the finger tip placed in the interclavicular notch and insinuated behind the manubrium recognizes aortal pulsation as a soft quiet impact. In marked hypertension a sense of sharp fling is often given to the examining finger. In luetic aortitis with aneurismal dilatation, there is a feeling of a broad, strong aortic impact, especially when decompensation is present; this is sometimes combined with a palpable thrill. The rough thrill accompanying aortic stenosis, and the sharp rise and subsequent collapse of the aortal walls in aortic regurgitation, can also be diagnosed by palpation in the jugulum.

Palpation over the base of the heart, on the right side, may be of great diagnostic importance. In extreme cases of rheumatic aortic stenosis a rough rasping systolic thrill is readily felt in the second and third right interspaces. The overacting aorta in aneurism and aneurismal dilatation of the first part of the aorta, and the sharp aortic distention in hypertension, are sometimes palpable over the same area. When present in the former, this probably denotes an extremely roughened, thickened aortal wall, or extreme dilatation. Bulging of the right anterior chest, found in adherent or erosive aneurisms, is obviously palpable. Aneurisms or aneurismal dilatation of the descending part of the arch, or even of the descending thoracic aorta, may occasionally be felt by placing the fingers snugly against the chest over the base of the heart, on the left side, or in the third and fourth left interspaces. In the latter position, the location of the impact area near the sternum, and the time of its appearance, serve to distinguish it from the impact due to ventricular systole (Chapter XIV).

In congenital pulmonary stenosis and in patent ductus arteriosus, thrills, usually systolic in time, are often palpable over the left basal region. In congenital patent interventricular septum, there is a rough, harsh thrill, systolic in time, palpable over the entire precordium, but especially over the lower sternum. In patients with dilated and over-acting pulmonary arteries, the sharp click-like closure of the valves can occasionally be felt by palpation over their site. This condition, combined with a murmur over the same area, is common in the mitral valvulitis in children, in whom it is sometimes incorrectly diagnosed as a congenital pulmonary lesion. Similar hyperaction is occasionally present as a temporary phenomenon in normal individuals.

Palpation in Mitral Lesions.—In typical mitral stenosis, with the heart beating rhythmically, there is a short presystolic thrill palpable in the apical region. When the auricles fibrillate and the ventricles beat irregularly and tumultuously, an intense and rough thrill, most prominent at the apex and occupying a varying part of the diastole, may be felt; if the irregular ventricular activity be controlled by digitalis, the thrill becomes less marked or entirely disappears. A palpable systolic thrill over the lower precordium not infrequently accompanies rheumatic regurgitant lesions, and may indeed be present when the murmur itself is faint or absent. This condition was found, for example, in a young girl without any previous rheumatic history; she had had frequent attacks of dyspnoea, and edema of the face and extremities. At the apex a scarcely perceptible murmur was heard; it was not transmitted. Over the lower precordium there was a very pronounced palpable thrill. Systolic precordial thrills may also be felt when the mitral cusps are thickened or covered with lime deposits as the result of a cardiosclerotic, non-rheumatic process (Chapter XII).

Further information regarding the **character of ventricular contraction** and of the **apex beat** can be gained by applying the flattened palm snugly over the lower precordial area. The booming broad ventricular shock and diffuse apical impact of hypertrophy may thus be readily diagnosed, particularly if the heart action is of normal rapidity or abnormally slow. Rapid action, even of a normal sized heart, usually gives the impression of enlargement, probably because of the rather violent impact against the chest wall. I have often corroborated the normal size of such hearts by fluoroscopic examinations. A somewhat thrill-like sensation is imparted to the examining hand during systole by violent ventricular overaction which usually accompanies tachycardia from any cause. Here the thrill may be due to sharp vibratory action of the auriculo-ventricular valves transmitted through the chest wall during violent ventricular systole. If the chest wall is of moderate thickness, it is sometimes possible to recognize the weakened impulse of an enlarged dilated heart by palpation. A double systolic apical impact, corresponding to the reduplicated first sound (so-called gallop rhythm, *q.v.*), may also be distinguished by palpation over the apex, especially when due to left ventricular hypertrophy, hypertension, or to aortic valvular lesions. The etiology of these reduplications will be discussed in connection with auscultation. Finally, *pulsus paradoxus* may be diagnosed by palpation, by the gradations of strength of ventricular contractions corresponding to the rhythmic waning and waxing of the pulse volume with inspiration and expiration. This is commonly attributed to pericarditis with effusion, or to intrathoracic tumors. I have observed it in occasional cases of myocarditis with severe decompensation, or in the agonal stage of cardiac failure.

PERCUSSION

To the ordinary and older methods of finger and pleximeter hammer percussion have been added the auscultatory, and, more recently, the flexed finger (Goldscheider's orthopercussion) methods.

The **auscultatory method** consists in placing the bell of the stethoscope over the center of the sternum and noting when scratch marks or light tappings made by the finger nail or finger tips first become audible. **Orthopercussion** consists in light perpendicular tapping over a sharply flexed finger held perpendicularly to the chest wall and acting as the pleximeter. These two methods were advanced as refinements by which the exact cardiac border could be more delicately and accurately outlined.

Inaccuracy of Percussion Methods. — Careful and continued use and observation of the above, as well as of the usual methods, checked by comparison with the unequivocal standards furnished by orthodiascopy and fluoroscopy, have convinced me that all methods of percussion are inaccurate and unreliable even for clinical purposes. Besides varying among themselves considerably in accuracy, there is no means of judging in advance which method will prove sufficiently exact in any individual case. In many instances, I have requested experienced and excellent clinicians to map out the cardiac border according to their own favorite method of percussion, and I have demonstrated to them by fluoroscopy that the results of their examination were rarely of sufficient exactness even for general bedside purposes. These statements apply to percussion for superficial dullness as well as for flatness. For example, very marked aortic dilatations were at times entirely overlooked; definite extension of the cardiac border to the right was not even approximately delimited. My own gross errors by the various methods of percussion have included practically all miscalculations: the delimitation of the size and of the upper border of the aorta, the right border of the heart, and the upper ventricular border. Figures 242 to 246 demonstrate some of these errors in patients with normal and abnormal hearts, in whom mistakes of outline varying from several centimeters to almost grotesque proportions have been committed. From the viewpoints of physics and acoustics, there seem to be good reasons for diagnostic errors in the use of percussion. Unless greatly enlarged and near the surface, the aorta, situated as it normally is mainly behind the sternum, is scarcely sufficiently thick-walled or with its contained blood of a sufficient density to cause differential dullness or flatness on percussion. Similar reasons make difficult the delimitation, by percussion, of the right cardiac border, consisting of the right auricle below and the great vessels above. Percussion of the left ventricular area is surrounded by less difficulties, for the ventricular mass is denser and more uniform; but its approximate size and contour — whether squatty, broad, erect, or gracile, subjects already discussed in connection with

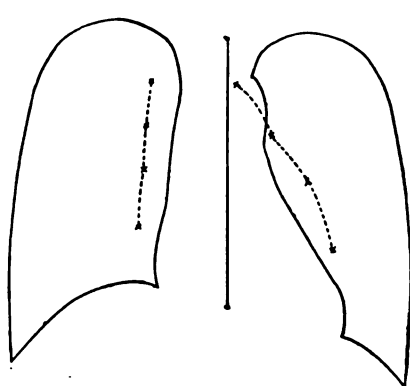


FIG. 242. — Normal heart — thin-chested male.

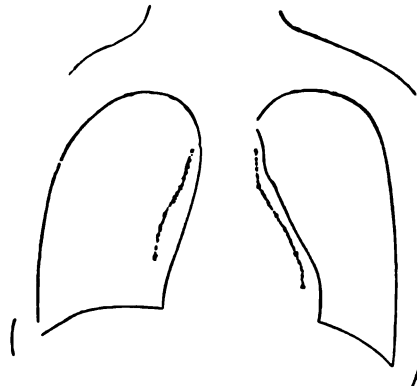


FIG. 243. — Normal heart — thick-chested male.

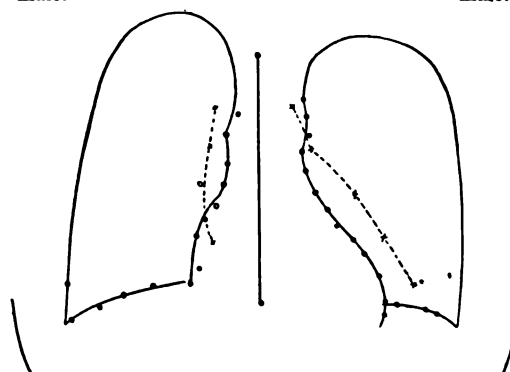


FIG. 244. — Normal heart — normal-chested male.

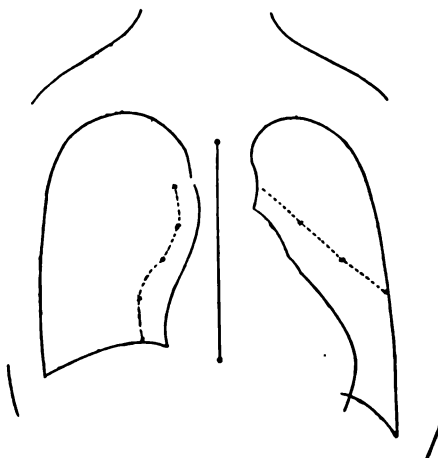


FIG. 245. — Mitral stenosis and regurgitation — normal-chested female.

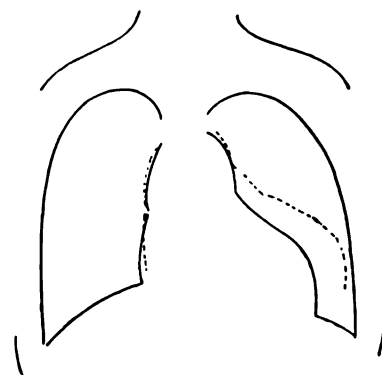


FIG. 246. — Aneurismal dilatation and left ventricular hypertrophy — thin-chested male.

FIGS. 242-246. — Difference in percussion (dotted lines) and orthodiascopic (solid lines) outlines of normal and abnormal hearts.

orthodiascopy (Chapter IX) — are considerations which I believe are beyond the physical limits of percussion. Frequent comparisons between percussion and radiographic findings have also convinced me that the left border of the heart may actually be several centimeters beyond the percussed outline, and that the lower point of the heart — the apex as viewed fluoroscopically at the end of inspiration — is usually from 2 to 5 cm. below its position as located by percussion or palpation. This difference between the 'palpable' and 'fluoroscopic' apex is especially noticeable in tall, lean individuals with flat chests, narrow elongated hearts, and mobile diaphragms.

A Rational Method for the Determination of the Cardiac Outline. —

In view of the limitations and of the inaccuracy of the percussion methods, I have attempted to apply the more exact knowledge gained by roentgenography to bedside study of the cardiac outline. The routine I use for this purpose is as follows: First, inspection, and then palpation of the precordium. Then auscultation to determine the presence and type of a possible cardiac lesion. Finally the mapping out of the cardiac outline. The initial step in the latter procedure consists in an attempt to determine the upper aortal border; this is accomplished by insinuating the tip of the finger in the jugulum. If the arch of the aorta is situated high in the thorax, it can be thus palpated; its corresponding upper limit is then marked on the chest wall. If the aorta cannot be palpated in this manner, it is considered as 'low.' Its approximate position may then be roughly determined by noting the most prominent point at which the aortic sounds are heard over the right base. This marks the probable position of the aortic valves; the upper aortal border lies about 2 or 3 cm. above this point. The next fixed point to be charted on the chest wall is that of the maximal apical impulse. In adults this is best determined by auscultation; in children, by palpation. As already stated, the actual position of the apex, as determined by fluoroscopy at the end of inspiration, is commonly 2 to 5 cm. below the point of maximal apical impulse; it varies with the amount of diaphragmatic excursion and the type of cardiac contour (Chapter IX). In most cases, these two variants can be estimated with some accuracy, even at the bedside, from the experience and knowledge gained by fluoroscopic examinations. Thus, in an individual with a normal heart, one variant — the type of cardiac contour — can often be roughly foretold by the configuration of the chest and extremities, and by the type of general physique. If an individual is thick-set, with short and pudgy hands, the heart is apt to be broad and flat, and the actual position of the apex will not be much below the point of maximal apical impulse. The lower right cardiac border (the right auricular border) is ordinarily from 3 to 5 cm. to the right of the midsternal line. If the individual is tall, spare, and thin-chested, with graceful extremities, the cardiac contour is usually long and narrow, the actual apex well below the palpable impulse, and the right auricular

border close to the right sternal margin. Exceptions in thin individuals are readily noted by the position of the apex. The second variant — diaphragmatic excursion — is more readily estimated. It depends upon lung expansion and upon the amount and thickness of the abdominal and chest walls. In obese patients with thick chest walls, diaphragmatic mobility is limited; the reverse is the case in well-knit persons with good lung expansion. Bearing these simple facts in mind, the true position of the apex may be readily and fairly accurately gauged. I had attempted to outline the remainder of the cardiac contour — the right aortal, and the borders of the pulmonary artery — by seeking the points over the right and left bases at which aortic and pulmonary sounds are heard most prominently. However, I have not found this method more accurate than merely drawing the cardiac outline of the normal heart by using the landmarks already determined — the upper aortal border, the calculated position of the actual apex, the right border, and the presumed type of cardiac contour, as guides for completing the outline. In this manner, in a series of normal hearts, I have in the majority of instances been able to gauge the cardiac outline with sufficient accuracy for clinical purposes, and have at any rate avoided any gross miscalculations, as shown by comparisons with the fluoroscopic examinations.

Determination of the Cardiac Outline in Cardiac Disease. — In cardiac disease, in addition to the above landmarks, further knowledge regarding the size of the heart is gained from palpation and auscultation; definite inferences may thus be derived regarding the probable shape as well as size of the heart. For example, in hypertension with an enlarged left ventricle and a booming apex beat, the lowermost part of the heart (the 'fluoroscopic apex') will be found considerably below the point of maximal apical impulse, and the cardiac outline will thus be found very much enlarged not only to the left but also downwards. In decompensated mitral lesions with diffuse apical impulse, especially with auricular fibrillation, the right auricular border will be considerably to the right of the midsternal line, and the 'actual apex' mainly to the left and not much below the usual position of the normal apex.

It is true that the method above described contains various inaccuracies, but I have found that careful balancing of the factors involved leads to closer and more reliable information than dependence upon any method of percussion. Perhaps further studies along these lines will yield more accurate data.

AUSCULTATION

Despite drawbacks and opportunities for error, auscultation is the most accurate of the four methods for the detection of the type of valvular or myocardial disease. The presence of **cardiac murmurs** (by which are meant **adventitious** sounds, not merely modifications of the

normal), their time relationship to events in the cardiac cycle, their intensity, quality, pitch, timbre, and the direction of their propagation over the precordium as well as through the larger arteries, the presence of audible thrills and rumbles, all require auscultation for their recognition. It is important to remember that murmurs may change in character with change in the patient's position. They are usually more intense when the patient is lying down; this may be because in other positions (standing or sitting) the apical impulse masks the murmur. Rapid heart action commonly intensifies the murmur of a mitral stenotic lesion although its duration is considerably diminished. For purposes of careful auscultation, one may attempt to slow the cardiac rate by having the patient breathe deeply and slowly; and to accelerate the heart action, by asking the patient to take a few rapid breaths. Another consideration which occasionally modifies both normal sounds and murmurs is the amount of pressure used when applying the stethoscope to the chest wall.

Valvular Murmurs in Rheumatic Endocarditis.—Organic valvular defects **do not always** produce murmurs. It is sometimes difficult from auscultation alone to determine which valve is affected, or to distinguish valvular from non-valvular affections. Other differential criteria will be discussed later. As further complicating factors in auscultation, the intensity and the propagation of the murmurs are considerably modified by the state of cardiac compensation, the amount of dilatation and hypertrophy, the rapidity of heart action, and the thickness of the chest wall.

Organic valvular murmurs are best grouped into those due to **narrowing** of the caliber of the valvular orifice (**stenotic lesions**) and those allowing **leakage** (**regurgitant lesions**).

A **mitral regurgitant** lesion produces a murmur systolic in time; the second pulmonic sound is often accentuated. The murmur may be soft and blowing in character, occupying only a part of the systole, and accompanying, not replacing, the first sound. When typical and well marked, the murmur is rough, low pitched, occupies the entire systole, replaces the first sound, is heard loudest in the apical region, and is transmitted laterally to the axilla. More rarely it is heard over the entire precordium. It is sometimes heard posteriorly between the angle of the left scapula and the spinal column, especially in children. The murmur may be accompanied by a palpable thrill. In decompensation with cyanosis a diastolic murmur transmitted downwards (the Graham-Steele) is occasionally heard at the second right interspace; it is probably due to dilatation of the pulmonary orifice with consequent relative insufficiency.

Mitral Stenosis.—In this lesion the second pulmonic is frequently accentuated as compared with the second aortic sound. The murmur itself is caused by auricular contraction forcing blood through a narrowed mitral orifice; the name auriculo-systolic given to this murmur many

years ago by Gairdner aptly describes its mechanism. It is characteristically a rough, rasping vibrant murmur; there is often a reduplicated second sound at the apex. The murmur seems crescendo in character, but electrocardiographic sound records tend to show that this 'crescendo' quality is only produced by its proximity to the loud first sound. The time of appearance of the murmur is immediately preceding systole (presystolic) or in late diastole. It may occupy the entire diastole. It is occasionally mid-diastolic, with a distinct gap between its completion and the following systole. This has been demonstrated by electrocardiographic sound records from patients in whom there was prolonged auriculo-ventricular conduction time (usually caused by digitalis); the murmur always occurred at the time of auricular systole, which in these cases bore varying relations to the diastole. A similar instance came under my observation — a young woman with mitral stenosis and gastric symptoms. Electrocardiographic tracings showed progressively prolonged *P-R* intervals (Chapter VII), so that at times there were independent auricular contractions; the rough, rasping murmur was always synchronous with auricular systoles, and, depending upon the conduction time, was heard in varying places in diastole. Such observations clinch the cause of the mitral stenotic murmur as due to auricular contractions. The mitral stenotic murmur often varies in intensity and duration. These variations probably depend upon the rate with which the blood flows from auricle to ventricle (Lewis), which in turn depends upon the difference in pressure existing in these two chambers. In the normal heart, at the beginning of ventricular diastole (and therefore of ventricular filling), the auricular pressure is high, the rate of flow more rapid. With the filling of the ventricle, the difference between ventricular and auricular pressure becomes less and less until the advent of auricular, which of course immediately precedes ventricular systole. At this time there is a sharp rise of auricular pressure and an **increased rate** of flow into the ventricle. Applying these facts to the slow, rhythmically beating heart with moderate mitral stenosis, the differential auriculo-ventricular pressure is manifest only at auricular systole, consequently the murmur with this type of rhythm is presystolic only. With more marked stenosis, or with more rapid heart action (shortened diastole), the differential pressure favors the auricle at the immediate completion of the ventricular systole; hence, here the murmur occurs not only in presystole but also in early diastole, or may occupy the entire diastolic period. It is especially in **auricular fibrillation** that the murmurs of mitral stenosis **undergo marked changes**. Absence of rhythmic auricular contraction precludes the usual presystolic element of the diastolic murmur. With comparatively slow and regular ventricular action, there is a distinct pause between the diastolic murmur and the following systole. The murmur may occupy early diastole alone and gradually wane because the auricular is then greater than the ventricular pressure and diminishes

with the filling of the ventricle. A rough, rasping diastolic murmur and a palpable thrill may lose these characteristics under the influence of digitalis and become much softer and scarcely audible. I believe this is because digitalis has produced steadier ventricular contraction with lessened differences in auriculo-ventricular pressure following ventricular systoles. When the ventricle beats very irregularly and rapidly, the murmur is short, sharp, and seemingly occupies the entire shortened diastolic period. With extremely rapid and tumultuous cardiac activity it may become barely audible or distinguishable, although in the occasional longer diastolic pauses its usual characteristics are evident.

Aortic Stenosis. — When typical, the murmur accompanying extreme aortic stenosis is the most marked of all the valvular lesions. It is then loud, rough, and vibrant, is systolic in time, is heard best in the second right interspace and is transmitted along the carotids. It is occasionally transmitted downward or posteriorly at the level of the third or fourth dorsal vertebra. The second aortic sound is often absent. This rough murmur is accompanied by a systolic thrill felt over the right base and carotids. The pulse wave may rise slowly (*pulsus tardus*, Chapter III), a characteristic sometimes recognizable by radial palpation.

Aortic Regurgitation. — This murmur varies considerably in intensity and in propagation. It is diastolic in time and may occupy only the beginning or the entire diastole. It usually replaces the second sound. The murmur is most often blowing in character; may be very soft, or, occasionally, quite loud and rough. The usual area of greatest intensity is at the right base; however, it is often heard loudest at the second and third left interspaces near the sternum. Not infrequently an aortic regurgitant murmur has exactly the characteristics of a mitral stenotic one; that is, it produces a presystolic sharp rumble at the apex (*Austin Flint murmur*). Under such circumstances, the regurgitant murmur at the base may be faint or entirely absent. Many theories have been advanced for the presence of this presystolic apical murmur of aortic regurgitation, but as yet no satisfactory explanation has been given. The consensus of opinion is that it is due to narrowing of the mitral orifice from the regurgitant stream of blood in the ventricle.

Tricuspid valvular lesions are very infrequently encountered. The etiology is rarely rheumatic, more often it is of streptococcic or luetic origin. Of the two types, regurgitant and stenotic, the former is the more common. The **regurgitant** lesion is characterized by a blowing systolic murmur, heard best at the lower sternum, and transmitted to the right; it is often accompanied by a pulsating liver and marked jugular pulsation. In one case that I observed, a distinct palpable thrill was also present. **Tricuspid stenosis** is exceedingly rare and has never been found as an isolated valvular lesion; it has been very in-

frequently diagnosed during life. The characteristic murmur is described as a presystolic rumble most prominent in midsternum or over the xiphoid. The lesion may be suspected in the presence of extreme cyanosis, right auricular hypertrophy, and marked pulsation in the veins of the neck and in the liver.

Valvular pulmonary insufficiency is exceedingly rare; it has never been observed as an isolated lesion and has been correctly diagnosed in only a very few instances. The murmur is described as blowing in character, diastolic in rhythm, heard best over the left second and third interspaces, and transmitted downward. It is usually accompanied by other endocardial murmurs.

Pulmonary stenosis commonly occurs as a congenital lesion. Its differentiation from aortic stenosis, which sometimes presents similar auscultatory signs, may be made from the fact that the murmur of the latter is propagated along the carotids, while the pulmonary stenotic murmur is transmitted downward.

A combination of lesions of the various valves, especially the aortic and mitral, is very common. The lesions in themselves tend to cause inorganic or so-called functional murmurs (*q.v.*) from the various degrees of valvular and ventricular dilatation they produce. In addition the valvular murmurs may not be typical; therefore it is sometimes difficult or even impossible to determine the exact valvular lesions that are present. The problem is further complicated by the frequent coexistence of pericarditis with its adventitious sounds. An extremely valuable guide in the differentiation of such complicated murmurs is careful comparative auscultation of the differing quality, pitch, and propagation of the murmurs. In this manner, the characteristic murmur of one of the valvular defects may be followed through an apparent maze produced by the presence of other valvular murmurs. Furthermore much information is gained by precordial palpation, which, with auscultation, may act as a guide in the determination of enlargements typical of the various valvular lesions.

MURMURS IN ATHEROSCLEROSIS

Besides rheumatic valvular affections, **atheromatous changes** in the mural endocardium, in the mitral valves, and in the aortic valves and walls frequently give rise to murmurs. The presence and significance of the murmurs due to such changes have not been sufficiently emphasized. Intraventricular murmurs, non-rheumatic in origin, are often present in cardiosclerosis. The cardiosclerotic process prevents coaptation of the mitral cusps and thus produces mitral regurgitation. This murmur is systolic in time and is heard best over the lower precordium; it is usually less loud than the rheumatic valvular mitral regurgitant murmur and is transmitted over a smaller area. Thicken-

ing and dilatation of the aortal walls, common in aortitis, give rise to a loud rough systolic murmur over the right base. As distinguished from aortic stenosis the murmur is less rough and vibrant, and only rarely propagated along the carotids. If aortic dilatation is marked, the systolic may be followed by a short diastolic murmur, due to regurgitation in the enlarged aortic cavity or to valvular insufficiency.

INTRACARDIAC MURMURS OF NON-ORGANIC ORIGIN

Murmurs arising in the heart and not due to organic disease have received many names and have been ascribed to various causes. They have been termed accidental murmurs, hemic murmurs, adventitious sounds, and functional murmurs. They have been ascribed to anemia, to relative valvular insufficiency allowing leakage and regurgitation of blood, to return of the blood in the large venous trunks. It is apparent that much confusion exists regarding both the terminology and the etiology of these intracardiac murmurs. In the description to be given, I shall apply the term 'functional murmur' to those adventitious cardiac sounds not produced by an organic lesion.

Functional murmurs are very common. The fact that they can occur without cardiac disease and without producing symptoms requires emphasis. They are often merely accidental discoveries in the routine physical examination. The presence of these murmurs in the healthy, as well as in such conditions as fever and anemia, suggests a varying etiology. The chief characteristics of the non-organic murmur are its softness and its limitations to small areas. There are numerous exceptions, however. The murmur may be rather loud, somewhat rough, and when found in the lower precordium, may be slightly transmitted above, or to the left. These exceptions are especially frequent in children and young adults. The most common site of the functional murmur is at the apex. It is systolic in time and accompanies the first sound, usually as a soft whiff. In addition to normal individuals, such functional murmurs are found in patients with rapid heart action, in those with flabby general musculature, and in those with anemia. In anemic individuals, the changed character of the blood is usually assumed as the etiological factor. This hypothesis, however, is not based upon any experimental data. In those with flabby musculature, I believe the cause to lie in dilatation of the musculature supporting the mitral ring, with consequent leakage and regurgitation; this is commonly termed **relative insufficiency of the mitral valves**. Perhaps many of the murmurs in the anemic (so-called hemic murmurs) heard in this location have the same etiology, for grave anemia conduces to a lack of proper muscular tone. In tachycardia, improper and faulty closure of the mitral cusps, alone or in addition to relative mitral insufficiency, may be the cause of the functional murmur at the apex.

A source of diagnostic confusion sometimes exists in differentiating the non-organic intracardiac murmur heard over the apex from the organic murmur of mitral insufficiency. The distinction between **typical** non-organic and **typical** organic mitral regurgitant murmurs is not difficult; the latter are rougher, louder, and are heard over larger areas than the former. The second pulmonic sound is apt to be accentuated as compared with the second aortic. When the characteristics of the two overlap, as, for example, in rheumatic fever in which the fever itself or the endocarditis may cause the murmur, the distinction between the organic and functional becomes exceedingly difficult. In doubtful cases with fever, examination must be made after the fever has run its course in order to arrive at definite conclusions. In anemia, the association with other 'hemic' murmurs, especially systolic murmurs at the right base, aids in the differentiation. The correlation of other data—lack of symptoms of heart disease, general type of muscular development, occupation (sedentary or otherwise), the type of heart as seen fluoroscopically—all these are elements which require consideration in the differential diagnosis between functional and organic murmurs. Non-organic murmurs are commonly met with in patients of sedentary habits, with flabby general musculature and ptosed abdominal organs. The distinction in the middle aged and elderly between a mitral non-organic murmur and that found associated with myocarditis is of extreme importance. The differentiation must be based upon the usual characteristics of the non-organic murmur. If it is soft, blowing, and not transmitted, one may infer that the mitral ring is dilated and that the murmur is of functional origin. The diagnosis of myocarditis must then be predicated upon the usual evidence of myocardial insufficiency, and upon the presence of an intraventricular murmur (*q.v.*) characteristic of atherosclerosis.

Aberrant fibers coursing through the ventricle, formerly termed aberrant tendons, can presumably give rise to loud systolic murmurs over the precordium. There seems to be no method of distinguishing these from other non-organic or organic murmurs. The tricuspid murmur of relative insufficiency may occur with decompensation from any cause of venous congestion: emphysema, myocarditis, endocarditis, etc. A systolic murmur of varying intensity is then heard over the lower sternum, usually transmitted to the right; an enlarged pulsating liver is occasionally present. Sometimes the liver pulsates in the absence of a tricuspid murmur. Since this could scarcely occur without regurgitation, it is clear that the latter may be present without producing a murmur. The frequency of relative tricuspid insufficiency without valvular disease is added evidence that mitral regurgitation with its murmurs may also be due to relative valvular insufficiency and to abnormal dilatation of the mitral ring. If tricuspid regurgitation and cardiac decompensation are extreme, the systolic murmur is occasion-

ally transmitted posteriorly on the right side between the mid-dorsal spine and the angle of the scapula.

A non-organic, soft, blowing systolic murmur is frequently heard over the aorta, particularly in anemia. It has none of the characteristics of aortic stenosis. This murmur is also found in decompensated mitral lesions. Its cause is not known. A short diastolic aortic murmur may be present in mitral lesions, possibly due to relative insufficiency from abnormal dilatation of the aortic ring.

Third Heart Sound. — In a large proportion of young normal adults, a faint third heart sound at the apex in mid-diastole may be heard by placing the patient in the left lateral position. It is soft and low pitched. Phonocardiograms of the sound have been obtained. The explanation given for the third heart sound is that it is due to the sudden floating up and tension of the auriculo-ventricular valves from the first onrush of blood from the auricle to the ventricle.

Extracardiac Non-organic Murmurs. — Pericarditis is naturally not included. Of non-organic murmurs of extracardiac origin, the most common is that termed the **cardio-respiratory** or **cardio-pulmonary**. It is usually soft and blowing in character, systolic in time, and heard best over the left base. Occasionally these characteristics change. The murmur can be rough and loud, and be transmitted along the left sternal border and lower precordium as a somewhat superficial, squeaky sound resembling a friction sound. The cardio-respiratory murmur is ascribed to compression of lung tissue between the heart and chest wall. During inspiration, the lung covers a large part of the root of the pulmonary artery and the upper precordial surface, facts which can readily be corroborated by fluoroscopic examination. This also explains why the murmur is usually heard best when the lung becomes inflated at the end of inspiration. However, it is sometimes best heard at the end of expiration, possibly because in some patients the pad of lung between the chest wall and the heart is abnormally large, thus diminishing the transmission of the murmur. The murmur is found frequently in children. It is not uncommon in frail women with thin chest walls. The fact that the intensity of the murmur depends upon respiratory phases and that it is never accompanied by a palpable thrill aids in distinguishing it from other types of organic cardiac murmurs heard in this area.

Reduplicated Sounds and Reduplicated Apical Impulses. — These reduplications have received various names, the most common being split sounds, gallop, canter, double, and triple rhythm, and "bruit de rappel." The term 'reduplication' seems preferable because, though many of its etiological factors are still obscure and problematical, it conveys some concrete idea of the underlying phenomenon.

Reduplicated Second Sounds are of **valvular origin** and can usually be clearly defined by auscultation. In the order of their frequency, reduplicated second sounds are heard at the apex alone, at the apex and

right base, at the right base alone, and lastly over the pulmonary artery. The time of their occurrence in the cardiac cycle and the study of electrocardiographic sound records demonstrate that they are due to closure of the semilunar valves. Their presence in various positions and combinations suggests differing etiology. In many instances conclusions regarding the cause can for the present be only tentative. One cause of reduplicated second sounds is **asynchronous closure** of the **pulmonic** and **aortic valves**. In this type the elements constituting the reduplication are heard very close to each other. In other types, the sound elements may be separated by a distinct hiatus. In these, the first part is undoubtedly due to closure of the semilunar valves, since it occurs in the cardiac cycle in early diastole soon after the *A-V* valves open. The second part of the reduplication has been ascribed to a process somewhat similar to the causation of the third heart sound, that is, to quick ventricular filling and resulting vibration of the floating cusps. This explanation, however, appears untenable, for the reduplicated elements seem identical on auscultation and have been so proven by graphic sound records. As a more plausible explanation, it appears to me that a sharp systolic wave can produce a secondary reflux wave which, impinging upon the already closed and tense semilunar valves, sets these in vibration and thus causes the second element of the reduplication. This would account for its frequent presence in tachycardia, in mitral stenosis, and over the great vessels at the base of the heart.

Reduplicated first sounds are usually classified under "presystolic gallop rhythm." I here include only reduplicated first sounds *without* reduplicated apical impulses, from which I believe they are etiologically distinct. Thus limited, reduplicated first sounds (without reduplicated apical impulses) are heard only in the apical region. It can be demonstrated by simultaneous electrocardiographic sound records and electrocardiograms that the **first element** of this type actually **lies in presystole**, and hence must be due to some presystolic event in the cardiac cycle. Most probably this is **auricular contraction**. The occurrence of the reduplication may depend upon a hypertrophied or overacting auricle.

Reduplicated Apical Impulse. — This type, always accompanied by a reduplicated first sound but of different origin from the above, is found especially in conjunction with hypertrophied left ventricle and in aortic disease. It is occasionally found in the overacting normal heart. Upon auscultation, besides the double first sound, there is a distinct sense of a **double shock or impulse**, often evident upon palpation alone. Since the reduplicated apical impulse has been found in lesions of the main branch of the auriculo-ventricular bundle, ventricular asynchronism is evidently one of its causes. The clinical diagnosis is aided by the fact that bundle lesions are usually accompanied by signs of myocarditis and by regular slow pulse rates between 50 and 60 per minute.

In addition to patients with bundle lesions, I have observed identical double apical impulses in those without electrocardiographic or other indication of bundle lesion. In several such instances there was definite fluoroscopic evidence that the double impact was due to a secondary ventricular movement following the primary systole. The exact cause of this double impact I could not definitely determine. In an effort to seek its explanation, the following must be recalled. It is known that the normal first sound is composed of three elements: first, tension of the auriculo-ventricular valves; secondly, the muscular element, ventricular contraction; and thirdly, ventricular impact against the chest wall. The first element of the reduplicated sound under discussion is probably due to the usual three normal factors above enumerated. Regarding the second part of the reduplication, it is difficult to state to what extent and proportion these three factors are concerned. To return to the fluoroscopic examination of patients with this reduplication, one may observe not only the ventricular systole, but what may be termed 'ventricular fling,' seemingly comprising different areas; sometimes this involves the apex alone, at others, the entire left ventricle seems to take part in the secondary movement. It is impossible to determine how far such ventricular fling resembles normal physiological systolic contraction. It may represent an attempt to regain ventricular tone which is interfered with by the distention produced by varying amounts of residual blood following systole. In general, it seems in harmony with clinical observations to seek mechanical intraventricular causes for the reduplicated apical impulse. A weakened myocardium may fail to properly empty the overdistended ventricle, a condition favorable to the production of a double impulse and reduplicated first sound. A similar condition can occur in patients with long-continued fever; as in typhoid, where myocardial weakness may be only temporary and functional. Again, it can occur in those suffering from myocarditis and resultant myocardial insufficiency. This was well illustrated in an instance of alcoholic myocarditis in which at necropsy the ventricles were found hypertrophied and riddled with scar tissue. A reduplicated apical impulse was present during the stage of decompensation alone; the electrocardiogram showed a deep M complex (Chapter IV). When compensation was temporarily restored, the double apical impulse and M complex disappeared, the ventricular complex assumed a normal outline.

Reduplicated apical impulses are frequently present in aortic regurgitation. In these cases they may be directly due to the blood which regurgitates into the ventricle; this then produces a sharp ventricular shock which becomes audible as well as palpable at the apex. A double apical impulse is also found in left ventricular hypertrophy with hypertension. The violence of ventricular action and the impact of residual blood may here account for the second element of the reduplication.

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CHAPTER X

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CHAPTER XI

ETIOLOGY OF ENDOCARDITIS AND OF CARDIOVASCULAR DISEASE

Classification. — In the following enumeration, aortic disease will be included because histologically its intima is a continuation of the endocardial endothelium, and disease affecting the one often affects the other.

Any classification adopted at this time must in a measure be arbitrary and tentative; for example, diseases now grouped as toxic alone may later be found to be of bacterial origin. Further studies may isolate specific factors in cases now grouped generically. With these limitations, the etiology of endocarditis and of cardiovascular disease may be conveniently tabulated as follows:

- | | | |
|---|---|--|
| 1. Chemical Agents | { | (a) Metallic Poisons.
(a) Alkohol.
(c) Tobacco.
(d) Diabetes.
(e) Gout.
(f) Products of Food Metabolism. |
| 2. Bacterial Agents | { | (a) Diphtheria — Pneumonia — Typhoid — Typhus.
(b) Rheumatism — Tonsillitis.
(c) Pyorrhœa — Mouth Infections.
(d) Pyogenic Abscesses. |
| 3. Bacterial Endocarditis. | | |
| 4. Spirochetal Infection — Cardiovascular Syphilis. | | |

1. CHEMICAL AGENTS

(a) **Metallic Poisons.** — Of these, the action of **lead** is the best known. This metal attacks the endocardium and aorta. In rare instances, the coronaries and their branches also become diseased. The renal arterioles are often involved and nephritis results. Depending upon the severity and the location of the arteriolar disease, the symptoms and physical signs may be those of aortitis, nephritis, coronary disease, or of endocarditis; or there may be a clinical picture combining these pathological entities in varying proportions. The patient's occupation, a history of colic, peripheral neuritis, the presence of a lead line on the gums, microscopic examination of the blood, and the chemical examina-

tion of the feces and of the total urine of several successive days for lead are data which are of aid in the search for lead as the etiological factor. Whether myocardial degeneration, when present, is primary or is secondary to disease of the coronary system is still an unsettled question. The selective action of lead on the arterial system seems to indicate that myocarditis results secondarily from the vascular disease.

Severe **phosphorus** poisoning produces fatty degeneration of the heart and of the arterial intima. **Other metallic** poisons — copper, mercury, arsenic — have a much greater selective action upon the gastro-intestinal canal, and upon nervous, osseous, and renal, rather than upon the cardiac structures. Except for the action of mercury upon the kidneys, it is questionable whether these metallic poisons produce cardiac damage that is clinically recognizable.

(b) The degree of damage to the cardiovascular system by **alcohol** is still a matter of dispute. Like other poisons, it affects the cardiovascular structures to a variable extent, so that necropsy reports showing absence of cardiovascular damage in chronic alcoholism are not necessarily evidence of its innocuousness. It is probably true, however, that the importance and frequency of alcohol as a cardiac poison have been overestimated. Alcohol attacks particularly the cardiac musculature; the result is myocardial disease varying from slight fatty degeneration to scar-tissue formation. The arterial system, when attacked, presents various grades of intimal thickening and calcareous deposits, and, in exceptional cases, degeneration of the remaining arterial coats.

The **clinical symptoms** referable to alcoholism are usually those of myocarditis and of myocardial insufficiency. The heart is moderately hypertrophied. As a clinical syndrome of moderate myocarditis may be cited that due to constant drinking of large amounts of beer (the "Münchener Bierherz"). In severe cases of alcoholic myocarditis, cardiac hypertrophy may be extreme. In one case that I observed at necropsy, a man of 45 who was an inveterate wine drinker, the ventricular musculature consisted mainly of scar tissue, the heart weighed twice the normal. Symptoms and clinical signs due to aortitis — an impure first and accentuated second sound at the right base, and hypertension — are also occasionally due to alcoholism.

(c) **Tobacco Poisoning.** — It is the general belief that tobacco users are particularly prone to aortic disease, coronary sclerosis, and myocardial degeneration, and that these pathological changes are caused by nicotine, the main tobacco alkaloid. This view is based chiefly upon the result of animal experimentation consisting in the injection of nicotine solutions in rabbits, with the production of a varying percentage of aortitis. The injections must be repeated at frequent intervals over a long period before aortitis results. As objections to inferences drawn from these experiments, it must be stated that others have found no abnormal aortal change following nicotine injections; indeed, one

observer found spontaneous changes in the aorta in a certain proportion of normal rabbits. Moreover, it is questionable whether these injections are comparable in their effects to the inhalation of tobacco smoke in man. For example, when the attempt was made to produce parallel conditions by forcing rabbits to inhale fumes on successive days from measured amounts of burning tobacco, no definite changes in the aorta were found. One observer who made a careful pathological study of the hearts of heavy smokers dying of various diseases found only a slight change in the papillary muscle; this he attributed to the rapid heart action usually found in smokers. From the experimental and pathological sides, therefore, it must be concluded that no incontrovertible proof has as yet been adduced that smoking in itself produces cardiovascular disease. On the other hand, as the result of physiological and pharmacological experiments, it is known that nicotine has a strong **neurotropic** action, particularly upon the sympathetic nervous system. Many of the symptoms complained of by patients with so-called 'tobacco hearts' — tachycardia, arrhythmias of various types (especially extrasystoles), syncope, precordial pains and distress — can be more readily and rationally explained, I believe, by the selective action of nicotine upon the sympathetic nerves and ganglia than upon the assumption of organic cardiovascular disease. In the majority of cases, when tobacco is withdrawn, cardiac irregularities and pain cease and patients examined months or years later show no evidence of organic damage to the heart or arteries. I believe, however, that if from any cause, a tendency to aortitis exists, its onset may occasionally be hastened by tabagism. Even this statement is only surmise, for it is not susceptible of clinical proof.

(d) **Diabetes** is frequently accompanied by arteriosclerosis, nephritis, and endocarditis, less frequently by myocarditis. These changes are presumed to be due to hyperglycemia and to foreign chemical substances in the blood which produce diminished alkalinity (so-called acidosis). The clinical symptoms and signs of the resultant cardiovascular disease, when not masked or complicated by diabetes itself, are high blood pressure, thickening of the palpable arteries, albumen and casts in the urine, and aortitis. The therapy should be directed to both diseases — diabetes and cardiosclerosis; relief or cure in the former is often followed by beneficial results in the latter.

(e) **Gout**. — Similar to diabetes, gout is also frequently accompanied by endocardial, vascular, and renal disease. An abnormal amount of uric acid in the blood and in the tissues may be the cause of these pathological changes, or both the latter and gout may be the expression of some common, as yet unknown, underlying factor or diathesis. The problem of therapy directed to the cardiorenal disease is rarely complicated by the presence of gout.

(f) **Products of Food Metabolism, Amino Acids, etc.** — The study of metabolism and of blood chemistry has shown the presence of amino-

acids and other protein derivatives in the blood. Experiments have demonstrated that protein feeding can produce changes in the cardiovascular and renal apparatus of animals. Though clinical proof is lacking, these investigations suggest that protein overfeeding in man may be followed by arterial disease, and that therefore proteins can act as a cardiovascular poison.

2. BACTERIAL AGENTS

In the production of disease, bacteria act directly, or by the elaboration of chemical poisons, *i.e.* of toxins. The latter are at present regarded as extremely complex chemical poisons of nitrogenous nature. It should be remembered that the various bacterial agents attack different parts of the cardiac structure and to a varying degree.

(a) **Diphtheria Toxins.** — In severe infections, marked destruction of the myocardium may result in death. In one instance, a girl of 10 with severe diphtheria and heart block, leucocytic infiltration in the junctional tissue and in the ventricular musculature was found. In another instance, a child developed heart block during the course of diphtheria. Electrocardiographic examination several years later still showed the arrhythmia; however, there were no cardiac symptoms. I have observed a child 8 years old with very toxic diphtheria, in whom, after a few days, there were attacks of convulsions coincident with slow rhythm. Polygraphic tracings showed regular radial and ventricular activity at the rate of 40 per minute; the venous tracing was unsatisfactory. Heart block was diagnosed. The child died one week later. Necropsy was not obtainable. From the virulent course of the disease, heart block seemed due to toxic degenerative myocarditis.

Milder toxic destruction of the myocardium is evidenced by the usual signs of myocardial insufficiency, especially by dyspnoea. Other evidence is the presence of arrhythmias:—heart block, auricular and ventricular extrasystoles, auricular fibrillation, tachycardia. These types may frequently interchange. They are apparently indicative of myocardial degeneration. Pathologically, the muscle cells undergo fatty degeneration, the valves and endocardium are rarely involved. With reference to the arrhythmias it must be remembered that, as in other acute febrile and infectious diseases, cardiac irregularities may occur in diphtheria at the onset or crisis, without any evidence of cardiovascular degeneration. Such irregularities at these times are probably the effect of toxins on the normal neurogenic cardiac control; they are harmless and rarely require medication. It is therefore important to differentiate the innocuous from the dangerous arrhythmias in diphtheria. With our present knowledge, this is best accomplished by a careful and complete examination of the entire cardiovascular apparatus, and not alone by a consideration of the types of arrhythmias. The presence of cardiac failure, slight or severe, — dyspnoea, cyanosis, edema, cardiac dilata-

tion—indicates that any type of irregularity, except physiological sinus arrhythmia, is of serious or even ominous import.

The study of the heart in diphtheria is further complicated by the occasional occurrence of unexpected, sudden death. This is usually regarded as due to cardiac paralysis. Some of these deaths can, I believe, be explained upon the assumption of a destructive, progressive myocardial degeneration with slight or no symptoms during life, or with the cardiac symptoms masked by those due to the diphtheria itself. In other cases, in which myocardial damage is not demonstrated at necropsy, these fatalities may possibly be regarded as anaphylactic phenomena affecting the cardio-inhibitory center or the intracardiac nerve supply.

Pneumonia.—In pneumonia pneumococci are not infrequently found in the circulating blood. The manner in which the myocardium is damaged is still in dispute. I am of the opinion that the myocardial damage is usually the result of toxemia. This is to some extent based upon the observation of two cases of heart block that occurred during pneumonia. One, a man of 70, came to necropsy. Macroscopical examination of the brain and spinal cord and microscopical examination of the heart, including the junctional tissues, showed that all these structures were normal. Another case, a man of 50, entered the hospital in collapse and semi-stupor. The temperature was 104°. There was pneumonic consolidation of the left upper lobe. The temperature ranged between 100° and 104° for one week. The heart sounds were scarcely audible, the radial pulse was regular, its rate between 100 and 120 per minute. At the end of one week, there was critical defervescence, the temperature fell to 97.4° per rectum, there was extreme collapse. The ventricular rate suddenly dropped to 44, and remained between 30 and 44 for four days. Although satisfactory graphic records could not be obtained, heart block was diagnosed by the clinical phenomena and pulse rate. The pneumonic area underwent gradual resolution. There were no general convulsions, but frequent convulsive tremors of the musculature of the upper and lower extremities. The stupor slowly deepened into coma. Suddenly, one week after the inception of the slow rhythm, the patient sat up in bed fully oriented, with good color, warm extremities, and a pulse and ventricular rate of 76 per minute. The heart sounds, though still faint, were readily heard. Except for two occasions a few days later, the pulse continued regular. From its occurrence with critical defervescence and its continuance during resolution, the cause of heart block seemed due to toxins liberated at the crisis and affecting the inhibitory cardiac control.

Action of Pneumonia on the Heart during the Acute Process and as a Late Sequela.—Endocarditis due directly to an invasion by pneumococci is by no means rare; this type will be discussed later. Many pathological examinations of the effect of pneumonia on the cardiac musculature have been reported. Slight degeneration (cloudy swelling)

has been the rule; occasionally more severe damage has been described. When widespread, these degenerative changes can undoubtedly cause death. But there is no evidence that slight cardiac degeneration, the usual change in pneumonia, is in itself sufficient to produce death; nor have attempts been made to correlate the amount and degree of pathological damage with the clinical or bacteriological evidence of toxicity. Experimental evidence pertaining to this subject indicates that pneumonia usually acts as a poison upon the functional power of the heart; for example, pneumonic blood perfused into a healthy dog's heart is immediately followed by very much weakened contractility. The contractions become normal when healthy blood is subsequently perfused. At present the conclusion seems warranted that death from pneumonia in the majority of cases is not due to demonstrable changes in the myocardium. On the other hand, damage to the cardiovascular apparatus as a late sequel of pneumonic infections has been insufficiently emphasized. Such sequelae produce symptoms only some months or years after the pneumonia has run its course, so that the connection between the two diseases is often entirely overlooked. In some instances the pneumonic poison seems to light up a dormant cardiovascular process; in others, it primarily produces this condition. Brief case reports of both types are herewith given:

Male, age 50, entered the hospital with a mild pneumonia. He recovered within one week. The only point of interest in a careful examination of the cardiovascular system was a slightly accentuated second sound over the right base, suggestive of aortitis. The Wassermann blood reaction was negative. The urine and the blood pressure were normal. Two months later the patient reentered the hospital with the history of a pneumonic attack three weeks before. He presented all the typical evidence of cardiosclerosis with decompensation: —anasarca, urine containing albumen and casts, high blood pressure, markedly accentuated second aortal sound. Evidences of the recent pneumonia were still present.

A physician, age 50, had for several years remarked occasional dyspnoea upon walking but never considered himself ill. Two years before coming under observation, he developed a severe pneumonia which ran a toxic course and lasted several weeks. A few months thereafter, cardiac symptoms began. He became dyspnoic and edematous. Hypertension developed. The urine contained albumen and casts. Orthodiascopic examination one year later showed marked enlargement of the aortic arch and of the left ventricle. With some remissions, the cardiac symptoms lasted until the time of his death, one year and a half later.

Both these cases illustrate the effect of pneumonia upon what seemed otherwise quiescent cardiovascular disease.

Male, aged 32, never drank or smoked. He had a grippe infection with fever six years previously; this lasted several months; pulmonary

tuberculosis was suspected but bacilli were never found. Cardionephritic symptoms began three years later. They were at first mild and consisted of occasional dyspnoea when climbing stairs. The signs of cardiosclerosis gradually progressed so that at the time of examination, although the patient complained only of slight dyspnoea and precordial pain, the left half of the chest was practically filled by a hugely hypertrophied heart. The systolic blood pressure ranged between 250 and 300 mm. of mercury, the first sounds at the right base and at the apex were exceedingly harsh and rough, indicative of probable calcification of the mitral valves and aorta. The second sound at the right base was markedly accentuated; the urine contained albumen and casts. The Wassermann blood reaction was negative. The clinical picture was that of extremely advanced cardiosclerosis and nephritis.

From the history and from the absence of other etiological factors, it is fair to conclude that the pulmonary infection six years before the onset of symptoms was the direct and only cause of the cardiovascular disease.

The action of diphtheria and of pneumonia as cardiovascular poisons has been described in some detail because similar observations apply in varying degrees to other infectious diseases. Typhoid fever deserves special mention in this connection. Therefore, careful search for such etiological factors should be made in every case of cardiac and arterial disease in which the cause is obscure.

(b) **Rheumatism — Tonsillitis.** — Of all diseases, **rheumatism** is the most frequent cause of valvular disease. Although the etiology of rheumatism is not as yet known, recent experimental research and clinical observation indicate that it is of bacterial, probably of streptococcic, origin. For the present, however, it is here classified under Toxemias. Valvular endocarditis is the most frequent sequel of a rheumatic infection. Myocarditis, which follows less frequently, usually occurs in the form of submiliary inflammatory nodes (Aschoff bodies). Permanent damage always results from rheumatic valvulitis and myocarditis; but here, as elsewhere in the body, if the infective process stops early, scar tissue may form before clinical signs of myocardial or valvular disease present themselves. The necropsy alone gives evidence of cardiac damage in such instances. Even if clinical manifestations are present, particularly in cases of mitral regurgitant lesions, the process may heal, so that valvular leakage stops and the patient remains clinically well. Such observations have been substantiated by post-mortem examination.

Tonsillitis is grouped with rheumatism in the etiology of cardiac disease, for it gives rise to joint manifestations and to endocarditis, similar to rheumatism. Even without joint involvement, tonsillitis frequently causes endocarditis. In fact, exceedingly mild and apparently harmless tonsillar and pharyngeal attacks, accompanied by

a few minute spots on the tonsils or pharynx, can be the source of an endocardial infection. For example, a vigorous young man of 21 developed mild pharyngeal grippe lasting three days; the highest temperature was 101.5° . Within one week typical signs of a mitral regurgitant lesion were present. Similarly so-called 'colds'—acute pharyngeal and nasal catarrh—can be the infectious nidus for endocarditis.

(c) **Pyorrhœa Alveolaris.**—Much experimental and clinical work has recently been done to show that the various organisms harbored in the mouth and producing pyorrhœa may, by systemic absorption, cause endocarditis and myocarditis. Among other bacteria, the streptococcus viridans has been especially accused. However, it has been shown that this organism is a fairly frequent inhabitant of the normal mouth; hence not only demonstration of the pyogenic organism is required but also definite clinical evidence that it bears an etiological relationship to the cardiac disease. In the face of negative blood cultures and in view of the other well-known causes for endocarditis, the correlation between pyorrhœa alveolaris and cardiac disease can only be established by a thorough examination of the mouth for carious teeth, for periostitis, and for pyorrhœa, and by radiographic examination of the roots of the teeth for evidence of absorption and caries. This constitutes the first step. Then a careful search of the entire body must be made for other possible infective foci which may cause cardiovascular disease. Cholecystitis, cystitis, deep-seated bone abscess, pyelitis are some of these bacterial conditions. So that even should roentgenography disclose root abscesses, and the mouth harbor pathogenic bacteria, it is for the clinician to decide from the clinical history, the type and probable duration of the cardiovascular disease, and from a complete examination for all other possible sources of infection, what weight, etiologically, should be given to the presence of root abscesses as the causative agent of endocarditis. Thus studied, it appears to me that the claims now made for the extremely frequent connection between oral infections and heart disease will be found unwarranted. This statement indeed fits in with general clinical experience. Very many children with valvular disease possessing healthy teeth have been observed as well as very many with carious teeth and normal hearts. In those patients with cardiovascular disease observed by me, in whom extraction of teeth had been done because of root abscesses, there was no effect upon the cardiovascular process or upon the clinical signs. One would not expect the small amount of toxins presumably elaborated and absorbed from a small dental focus to be a *frequent* cause of endocarditis; one would expect sufficient anti-bodies to be developed to prevent cardiac mischief in the great majority of cases. On the other hand, it cannot be denied that, in an extremely susceptible individual, general systemic damage and cardiac disease can thus occasionally, though I believe only very

rarely, be produced. It is, therefore, necessary for the *clinician* to weigh all factors and not to conclude too hastily that because pus, pathological bacteria, or small root abscesses are found in the mouth they are necessarily the cause of the cardiovascular disease in that individual. We are greatly indebted to the dental profession for indicating the teeth as a cause of endocarditis, but the physician must finally judge all the evidence in every case before he can decide whether the cause of endocarditis lies in the mouth or elsewhere. None of the statements here made of course militate against the fact that purulent mouth conditions should be carefully treated, or that children with diseased teeth should receive proper dental attention in order to remove that source of a possible endocarditis. My main object has been to refute the now widely accepted opinion that diseased teeth *commonly* cause disease of the heart. In those exceptional cases under my observation in which alveolar pyorrhœa was apparently the cause of the existent cardiovascular disease, the pyorrhœa was frank and usually severe even to the time that the patients presented themselves for examination because of cardiac symptoms. There was usually a long history of foul breath and of decaying and loose teeth. The patients were of middle age or past middle age. When brought out by questioning, the history of pyorrhœa was definite and of several years' duration. Of extreme interest and importance is the fact that, symptomatically and pathologically, the picture is usually that of cardiosclerosis (*q.v.*). It seems as if the toxin acts primarily upon the arterioles of the cardiovascular system, affecting in varying degrees the vessels of the heart and kidneys. Its final effect upon these organs often closely resembles, indeed sometimes coincides with, the pathological picture of senile cardiosclerosis (*q.v.*).

(d) **Pyogenic Abscesses.**—These may occasionally produce endocarditis by the production of a bacteremia or from toxins. In endocarditis, where a frank etiological factor cannot be found, careful search should be made for hidden abscesses as possible causes.

3. BACTERIAL ENDOCARDITIS

Bacteria were demonstrated in valvular vegetations by Heiberg as early as 1869. Since then, many types of organisms have been recovered from such vegetations, and, more recently, have been isolated from the blood.

The question of systemic bacterial infection in rheumatic fever is still unsettled, although, as already indicated, there are good clinical and experimental grounds for considering it a bacterial disease. The organisms found in the blood which produce endocarditis are grouped, according to Simons' modification of Litten's classification, as follows:

1. Ordinary Streptococcus.
2. Small Streptococcus. (Probably all alike, but variously named by different observers.)

{	Streptococcus viridans (Schottmüller). Endocarditis cocci (Libman). Modified pneumococci (Rosenow). Saprophytic Streptococci (Horder). Streptococcus tenuans (Hastings).
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3. Staphylococcus albus and aureus.
4. Pneumococcus.
5. Gonococcus.
6. Meningococcus.
7. Bac. coli.
8. Bac. influenzæ.
9. Bac. pyocyaneus.

From the statistics of various authors, the most common invaders in **acute** bacterial infections are, in the order of their frequency, the streptococcus pyogenes and the staphylococcus aureus; fairly frequent are the pneumococcus and gonococcus; the streptococcus viridans is very rare. The other bacteria — staphylococci, meningococci, bac. coli, bac. pyocyaneus, and bac. aerog. capsulat. — are only occasionally found. The **chronic** invaders are, in the order of frequency, the streptococcus viridans (which is very common), the streptococcus pyogenes, pneumococcus, bac. influenzæ, and the gonococcus. It is thus seen that the gram-positive chain cocci are the chief causes of bacterial endocarditis.

Relative Frequency of the Bacterial Invasions and of the Valvular Affections. — The mitral is the valve most frequently affected alone; next in frequency is the aortic. Bacterial infection of the pulmonary or tricuspid valves alone is exceedingly rare. The most common combination of a multiple infection is the mitral and aortic; the next, the tricuspid and mitral. Vegetations on the walls of the auricle and ventricle have also been described, but they do not occur without vegetations on the valves as well. The pathological features of chronic streptococcus viridans are discussed in connection with the clinical features of that affection (*q.v.*).

4. SPIROCHETAL INFECTION

The immense strides made possible in the detection of syphilis by the discovery of the causative organism (*spirocheta pallida*), and by the use of the Wassermann reaction, have enhanced our knowledge of the frequency of lues as a cause of cardiac disease. The relation of syphilis to valvular disease, especially to disease of the aorta, although not proven, had long been suspected. Syphilis has now been demonstrated as a very common cause of disease of the heart muscle; in fact, myocarditis is occasionally present in the secondary, as well as in the tertiary, stages of lues. By special staining methods, the spirochete has been found in the aorta and in the cardiac musculature. The most marked, and probably the earliest, pathological involvement consists in a periarteritis

of the arterioles of the coronary system, resulting in various types of muscle degeneration (fibrosis, fatty degeneration, brown atrophy). As a later involvement, gumma and gummatous infiltrations also occur. The valves and walls of the aorta are the most common situation for cardiovascular syphilis. The picture varies from mild arteriosclerosis to extreme cicatrization with calcification. Thickening of the mitral cusps is less frequent. It may, however, be sufficient to produce signs of a mitral regurgitant lesion. Endocardial changes varying from slight opacities to extensive degeneration may coexist.

CHAPTER XII

PATHOLOGY OF THE ENDOCARDIUM AND MYOCARDIUM, AND OF CARDIOSCLEROSIS

Structure of the Endocardium. — The endocardium is composed of connective tissue containing smooth muscle- and elastic fibers; its free surface is covered by a layer of endothelial cells. Underneath this layer is one of loose areolar tissue continuous with the intermuscular connective tissue septa; this stratum also contains the blood vessels. The cardiac valves are composed of fibrous connective tissue covered by endocardium; they contain muscle fibers at their attached margins. The normal semilunar valves have no blood vessels; the normal tricuspid and mitral valves contain blood vessels only in the muscular tissue at their bases, the muscular tissue being a reflected layer of varying size from the main ventricular mass.

In the description of the different pathological varieties of endo- and myocardial changes, the classification given by E. Kaufmann will in the main be followed.

The **endocardium** is subject to the following **degenerative changes**:

Fatty Degeneration. — Macroscopically, pure or yellowish white areas are found in the endocardium, especially on the mitral valves in old people. Such degenerative areas usually represent senile changes; they are, however, occasionally the result of anemia, toxemias, and infections.

Sclerosis. — The affected endocardium takes on a white, thickened, and hyaline appearance. An entire valve, especially the mitral, its free border, or parts of the mural endocardium, may be involved; in addition to these changes there may be calcareous deposits. Occasionally the sclerotic areas undergo mucoid degeneration. Microscopically, the sclerotic patches are found to consist of strands of connective tissue which have undergone hyaline degeneration.

Atheromatous Degeneration. — This consists of focal necrosis in the valvular tissue and may include any of the changes already mentioned.

The above degenerative types are as a rule found in elderly people, and may be regarded as senile in nature. They are occasionally encoun-

tered in the middle aged, very rarely in the young. The valves especially attacked by these degenerative processes are those previously inflamed. The result is that the free surface of the valvular endocardium becomes roughened; adherent thrombi may form which later, following a slow productive inflammation, become partially or totally organized. Thickened patches are thus formed. The end result of all these changes is an irregular, more or less diffuse, sclerotic, and atheromatous thickening of the valves, with consequent profound change in their structure, shape, and function. They can no longer close the cardiac orifices properly, thus giving rise to various abnormal blood currents and to murmurs (Chapter X).

It is at times difficult to differentiate this primary sclerosis and atheroma from chronic fibrous and infectious endocarditis.

Endocarditis, sometimes called thrombo-endocarditis because of its chief pathological feature, is an inflammation involving the valves and occasionally also the mural endocardium and chordæ tendineæ. As discussed in another connection (Chapter XI) endocarditis may be produced by bacteria, their toxins, or by foreign chemical substances flowing in the general circulation. In bacterial endocarditis (*i.e.* where quantities of bacteria are found in the circulating blood) the usual sequence of the pathological process affecting the valves is as follows: The bacteria form clump-like deposits which appear as very fine grayish granules. Beneath these, there is destruction and necrosis of the endothelial, and finally of the deeper layers of the valvular structure. The extent of the destructive process apparently depends upon the virulence of the invading bacteria. If the endothelial layer is not entirely destroyed, it shows only cloudy swelling. Thrombi from the circulating blood, consisting of platelets, fibrin, and white and red blood cells, become adherent to the damaged surface. With the washing off of the bacterial clumps by the blood stream there remains a small defect with a necrotic base, to which thrombi again become attached. Where blood vessels are present in the valves, an exudative inflammation surrounds the necrotic areas. Where the vascular supply is absent there is a growth of connective tissue cells and an increase in lymphocytes; later, there may be formation of new blood vessels. In **acute foudroyant cases**, also called **ulcerative, malignant, or septic** endocarditis, in which there is extreme bacterial virulence with large bacterial deposits on the valves, the necrosis spreads to the deeper layers. This is due to the primary effect of the bacteria or their toxins, or is secondary to an intense purulent inflammation. In this manner, large valvular defects are soon produced. In **less virulent subacute and chronic cases**, there is connective tissue production of flat wart-like excrescences upon the valves. This process has been variously termed **verrucous, productive, simple, or rheumatic** endocarditis. The damage is occasionally so mild and superficial that the only sign of a previous inflammation is a small amount of scar tissue. Since the verrucous and ulcerative

varieties of endocarditis are not always distinct, and may merge into each other, they may both occur together as a mixed process. A shrinkage of the granulomatous connective tissue produces what is known as **chronic fibrosis** or **fibroplastic** endocarditis.

Verrucous endocarditis is usually found in the left heart in adults. The ulcerative type is seen fairly frequently in the right side of the heart. In the new born, the site of predilection for verrucous endocarditis is also the right heart.

The verrucæ and the formation of fibrous tissue give rise to stenoses of the valvular orifices (Stenotic lesions, Chapter X), or the valves no longer properly close the orifices, they become incompetent and allow the blood to flow back into abnormal cardiac chambers (Regurgitant lesions, Chapter X).

It is of importance to correlate, where possible, the pathological picture of endocarditis with valvular disease as encountered clinically. Thus, in **acute endocarditis**, the usual change consists in the production of ulcers with thickening of the cusps, and the presence of healed areas and of granulations. Valvular deformity is slight or absent. In the longer continued, **subacute cases** of endocarditis, verrucæ along the margins of the valves represent the prominent pathological change. Valvular deformity is moderate. In **chronic endocarditis**, there is production of new tissue affecting not only the valves but also the chordæ tendinæ; the latter become stiff, the valves hard and inelastic. The result of both of these changes is marked deformity; in the case of the mitral valves, there is lengthening and funnel-like formation of the mitral opening, accompanied by varying degrees of stenosis.

MYOCARDIUM

The following are the chief pathological changes occurring in the myocardium.

Simple and Brown Atrophy. — In both, the cardiac fibers become smaller and the entire heart is decreased in size. In addition, in brown atrophy pigment is found as small granules in the sarcoplasm. The entire heart presents a brownish appearance. Both types occur in inanition, and in various cachectic states. Brown atrophy especially is regarded as a senile change.

Parenchymatous Degeneration. — Microscopically, this is marked by cloudy swelling. Macroscopically, the cardiac musculature has an opaque, dark red, somewhat spotted appearance, and is softer and more friable than the normal muscle. This degenerative change is a frequent accompaniment of infectious fevers and of biological and chemical poisons. It is also found in severe anemia.

Fatty Degeneration. — This exists as a primary process or represents a later development of parenchymatous degeneration. Depending upon its severity, the myocardium is studded with fat droplets of

various size. Macroscopically, the musculature shows patchy or diffuse yellowish areas. When the damage is extreme, the muscle is flabby, friable, and grayish in color. Very many factors, especially those that alter the quantity and quality of the blood, may cause fatty degeneration. For example, it is found in severe primary anemias, in anemia following hemorrhage, in infectious diseases, in chemical intoxications (especially from phosphorus, arsenic and alcohol), in coronary disease, in cardiac hypertrophy, and in chronic nephritis.

The "Fatty" Heart. — Fatty degeneration must be distinguished from what is ordinarily termed the "fatty heart." In the latter the organ is covered by more or less extensive fat pads. Sometimes these lipomatous masses insinuate themselves between muscle bundles and appear as fat clumps or spots under the endocardium. "Fatty hearts" are usually the accompaniment of fat accumulations in other parts of the body, in obese and thickset individuals; very rarely these masses are found as secondary changes in the cardiac atrophy of cachexia.

Disturbances of the circulation sometimes give rise to myocardial changes. This is especially true of focal anemia subsequent to interference with the coronary circulation from emboli in the main coronaries or their subsidiaries, or from coronary endarteritis. Either of these impedes or entirely stops the intrinsic arterial supply. Coronary endarteritis causes extreme intimal thickening or calcareous deposits; thrombi may become adherent to the denuded roughened intimal surface.

Anemic Necrosis. — Following complete obliteration of one of the smaller coronaries, the corresponding cardiac area becomes ischemic and pale, and undergoes coagulation necrosis, a process sometimes termed anemic necrosis or anemic infarct. The infarcted area may become myomalacious. In rare instances, such softened areas develop into aneurisms of the cardiac wall which may finally rupture. A more frequent outcome is organization of the anemic infarct; the necrotic material is absorbed, and vascularized granulation tissue takes its place. This ends in fibrous or fibroplastic myocarditis, and, finally, in scar tissue formation. If the process is widespread, the ventricular musculature appears striped on section. It presents a somewhat checkered appearance if older scars are found in conjunction with fresh necrotic areas.

Acute Interstitial Myocarditis. — As the result of bacterial infection, in which a clump forms the inflammatory nidus, an area of cloudy swelling, necrosis, and fatty degeneration of the musculature is developed. The necrotic area becomes infiltrated by a zone of leucocytes. If the leucocytes penetrate the necrotic zone, an abscess results. These abscesses are usually minute, though by confluence they may sometimes be readily seen by the naked eye. Small abscesses end in scar tissue formation by destruction of bacteria and liquefaction and absorption of the pus cells. The defect is then covered by granulation tissue coming from the surrounding musculature. On the other hand, these

foci may cause purulent pericarditis, emboli, ulcerative myocarditis, and cardiac aneurisms. Any of these sequelæ can directly or indirectly cause death. In those instances in which the inflammation stops short of abscess formation, the process may end in the production of granulation, of young connective and, finally, of scar tissue. This occurs especially in the myocarditis accompanying rheumatic endocarditis in which large-celled infiltrations (Aschoff's bodies) are found. The cells comprising these infiltrations are probably derived from wandering mononuclears and not from muscle or connective tissue.

Chronic Fibrous Interstitial Myocarditis.—This is a secondary productive process resulting in the formation of fibrous areas containing elastic and connective tissue. It may follow acute myocarditis, parietal endo-myocarditis, coagulation necrosis of the cardiac muscle, or cardiac abscess.

CARDIOSCLEROSIS

Of late there have appeared in the literature and clinical medicine the generic terms cardiosclerosis, cardiovascular disease, and cardio-renal disease. These are convenient though somewhat inexact terms which are meant to describe and group certain clinical conditions. There is no single pathological picture which accurately describes these groups. As the names denote, the pathological change in the myocardium and endocardium is an exceedingly variable one. It depends upon the amount and type of the other concomitant diseases affecting the kidneys and general arterial structure. The usual underlying factor is a general degenerative change of the entire vascular system, but especially of the smaller arterioles (the capillary fibrosis of Jores) of the heart and kidneys. The heart then presents changes corresponding to this widespread cause, in addition to changes wrought by renal disease. The heart is either atrophic, with a thickened, sclerosed, and calcareous endocardium and indurated scars in the myocardium; or it is enlarged, the coronaries showing signs of mild or widespread disease varying from slight thickening to extreme calcification and obliteration of the arterial lumen. This process may involve the smallest coronary branches. The aorta is dilated, its intima thickened and atheromatous, containing calcareous deposits. A similar process affects the semilunar valves which, as a consequence, become incompetent or stenotic. The ventricular endocardium shows various patches of fibrotic thickening. Similarly, the mitral valves are infiltrated by fibrous patches on their surface or along their free margins. The valves may even be incrustated with calcareous deposits. The myocardium presents indurated areas of scar tissue formation, or the entire musculature is riddled by massive formations of this character. Engrafted upon these commoner changes are those which frequently accompany hypertension and renal disease, *i.e.* massive hypertrophy usually of the left but sometimes of both ventricles.

CHAPTER XIII

CLINICAL PHENOMENA AND SYMPTOMATOLOGY OF ENDOCARDITIS

For clinical purposes, I shall divide valvular endocarditis into the **rheumatic**, the **bacterial**, and the **chronic streptococcus viridans**. Cardiac syphilis and general cardiosclerosis are discussed separately.

RHEUMATIC ENDOCARDITIS

Clinical phenomena accompanying acute endocarditis may be divided into the general **rheumatic** and the **endocarditic** manifestations. The former include joint and muscular symptoms, chorea, tonsillitis, erythema nodosum, subcutaneous fibroid nodules, etc. These rheumatic manifestations do not always run parallel with the degree of endocardial involvement: the rheumatism may be mild, the endocarditis severe, or the reverse. I have, for example, noted the appearance of severe infection of the mitral valve following very mild catarrhal invasions of the upper air passages. One instance was that of a young, vigorous adult of twenty, suffering from mild pharyngeal catarrh. His highest temperature was 100.4°. After three days, a typical mitral lesion developed.

Endocarditic manifestations depend upon physical signs, and upon the type, course, and complications of the valvular infection. The physical signs of the various lesions are described in a separate chapter (Chapter X). Writers have classified endocarditis according to special features (fever, cerebral complications, etc.), the assumed pathological process (verrucous, warty and papillary endocarditis), or the results of blood culture. Since the various groups merge, and bacterial invasions (*e.g.* streptococcus viridans) may complicate rheumatic endocarditis, sharp divisions are purely arbitrary and cannot be maintained. I purpose therefore to describe the usual clinical type of simple rheumatic endocarditis, with such variations as are important.

The one prominent symptom of **acute rheumatic endocarditis** is fever. When the general rheumatic manifestations are active, it may be impossible to trace the source of the fever, for it may then be due to rheumatism or endocarditis. When the rheumatic course is prac-

tically afebrile, any sharp rise of temperature, especially if sustained, is suggestive of an endocardial involvement. Fever continuing after recession of rheumatic signs in the joints and elsewhere is strongly significant of endocarditis. Fever may be so slight that the temperature may have to be taken frequently in order to establish its presence. This is especially true in the mild type of acute endocarditis of adults. More often there is a rise of temperature of one or two degrees; its usual accompaniments — lassitude, anorexia, and headache — are then present. Hyperpyrexia is uncommon. An initial chill or rigor is rare. It occasionally happens that there is no rise of temperature during the entire course of the endocarditis; this of course does not preclude the possibility of fever before the patient came under observation.

Certain **prodromal symptoms** suggestive of endocarditis may exist, particularly in children, even in the absence of fever. Such children, otherwise robust and healthy, complain of feeling tired, and become anemic and less active. These manifestations have no reference to the heart, for the children are not dyspnoic, and physical signs of endocarditis are not found. It is only when mild rheumatic symptoms, such as fleeting pains in the limbs, occur, that a slight rise of temperature for a few hours may be observed; with it, the physical signs of endocarditis often become evident.

Between the very mild, almost non-febrile cases, and the commoner ones with moderate temperature, there exist all grades and types of febrile disturbances.

The **physical signs** of **acute** endocarditis are inconstant; they usually vary with the intensity of the process. The latter may be slow and gradual, so that it is only after a long time that frank physical signs of valvulitis appear. On the other hand, physical signs, especially in children, may be immediate and unmistakable. Once established, the auscultatory phenomena of the valvular affections are those already described (Chapter X).

Other Symptoms and Signs of Acute Endocarditis. — The first hint of endocardial damage may be slight acceleration of the pulse. In adults, the rate may reach 100, in children, 120 per minute. This pulse acceleration is commoner in children than in adults. It may precede the physical signs of endocarditis by several days. This occurred in a young woman of twenty-five with articular rheumatism and without temperature, who suddenly developed moderate tachycardia; several days after rheumatic manifestations had ceased, the physical signs of endocarditis were present. Next in frequency to moderate pulse acceleration are extrasystoles. Paroxysmal tachycardia, auricular fibrillation, and flutter are uncommon. Prolonged conduction time is sometimes found; it is regarded as an evidence of myocardial rather than of endocardial involvement. In very rare instances myocardial involvement shows itself by atrio-ventricular heart block.

Subjective symptoms of various kinds are sometimes referred to the precordium during the course of acute endocarditis. They consist of indefinite sensations of weight or pressure on the chest. Occasionally "thumping" sensations are complained of, although the heart rate is normal; this seems due to abnormally strong ventricular contractions. There are at times "sticking" or "stitch-like" precordial pains (Chapter XXI). Pain may be present not only over the precordium, but may also radiate to the left shoulder and neck and to the left intercostal spaces. In general, the pains are not severe; they are more common when tachycardia is present.

Cardiac decompensation in any of its phases is very rare in simple acute rheumatic endocarditis.

To summarize: fever, anemia, arrhythmias, or precordial sensations constitute suggestive signs of the onset of acute rheumatic endocarditis. Definite physical signs of a valvular lesion are necessary in order to clinch the diagnosis.

Exacerbations of simple rheumatic endocarditis are, in general, marked by signs and symptoms similar to the original onset of the disease. Rheumatism, pronounced or obscure, is often present. In some respects, however, rheumatic endocarditic recrudescences differ from the primary attack. For example, pulse irregularities are more frequent. Tachycardia — simple pulse acceleration or the paroxysmal type — is particularly common. Extrasystoles occur next in frequency. Auricular fibrillation, present throughout the entire exacerbation or coming in attacks, is by no means rare; in this respect, especially, exacerbations differ from the original onset. Auricular flutter is occasionally observed. Subjective precordial sensations are more common; they vary from sensations of pressure to frank attacks of precordial distress. The physical signs of the valvular lesion are more pronounced unless they are masked by rapid or irregular heart action. Mild disturbances of compensation — slight edema of the legs, enlarged liver, dyspnoea, etc., begin to make their appearance.

CHRONIC ENDOCARDITIS

Depending upon the duration and frequency of rheumatic exacerbations, the symptoms merge into those of chronic endocarditis, the group embracing the great majority of cases of valvular heart disease. Patients with chronic endocarditis and perfect compensation may continue in good health for years; cardiac symptoms may never occur. These are the fortunate instances in which the disease has become quiescent and the cardiac damage has not been extreme.

The chief **symptoms** of chronic rheumatic endocarditis are due to myocardial insufficiency and to decompensation. Brief reference must here be made to the underlying cardio-muscular cause of decompensation. The heart, like voluntary muscles, possesses the property of

shortening during, and of lengthening after, contraction. In health, this function is perfect, and permits without damage a considerable degree of dilatation (*e.g.* after exercise). In other words, it is this ability to contract and expand which fundamentally indicates what is known as the 'cardiac reserve.' As the result of valvular disease there is a varying amount of change in the ventricles and their chambers; this consists in hypertrophy, dilatation, and myocarditis. These abnormal factors compromise to a varying degree the underlying function of lengthening and shortening of the cardiac fibers, and hence interfere with the reserve power of the heart. In consequence, the cardiac power is decreased, and signs of decompensation appear. There is also evidence that differences in tone may play a part in the symptomatology. For example, patients may complain of dyspnoea, of vaso-motor symptoms, and even of precordial distress without any physical or clinical evidence of cardiac dilatation. These symptoms may appear suddenly, remain for a few hours or a day, then disappear without signs of decompensation.

Dyspnoea. — Our knowledge of the cause of dyspnoea has been advanced through recent chemical studies of the blood. Abnormal products or abnormal amounts of normal products producing so-called acidosis have been found. Experiments show that alkalies injected into the blood stream cause apnoea, and that very small quantities of acids and of acid salts, similarly injected, produce hyperpnoea. Carbon dioxide acts chemically in this respect like a mineral or organic acid; it causes an increase of the hydrogen ions in the blood, the factor which measures 'acidosis.' The respiratory center is quite sensitive to changes in blood reaction. The mechanism of its regulation is as follows: acidosis (diminished blood alkalinity) stimulates the respiratory center to increased activity; there is a consequent reduction in carbon dioxide, thus tending to keep the hydrogen ion concentration of the blood at the normal level.

There are several methods of estimating the carbon dioxide content of the blood. It may be measured directly by examining blood withdrawn from a vein (Van Slyke method); or it may be estimated indirectly by various apparatuses for determining the carbon dioxide content and tension of the alveolar air. The results derived from these two methods run fairly parallel. Conclusions drawn from such examinations bear an important relation to cardiac dyspnoea, as will soon be shown.

Cyanosis. — It is a common clinical observation that cyanosis, often a prominent sign in valvular disease, is not necessarily accompanied by marked dyspnoea. On the other hand, patients of the cardio-sclerotic (*q.v.*) type, especially those with renal complications, are very apt to be dyspnoic. They are frequently not cyanotic; indeed, they may be decidedly pale. The dyspnoea is often nocturnal. It is thus evident that dyspnoea is present in two different groups of cardiac patients: those who are pale and those with cyanosis. In the cyanotic

group, blood examination shows no abnormal increase of non-volatile acids; the carbon dioxide tension of the alveolar air is usually normal; there is no or only slight decrease of blood alkalinity. In the **anemic** group, renal complications are frequent, the blood pressure often high, and venous congestion is, as a rule, absent; the carbon dioxide tension of the blood and of the alveolar air is decreased, the acid products increased (acidosis); both changes may be extremely marked. Such patients often have uremic manifestations. At present, the belief is that these abnormal products in the blood are due, not to overproduction, but to retention from renal insufficiency.

Cyanosis, with no or only slight dyspnoea, is most strikingly seen in chronic uncomplicated valvular disease. Unless renal complications are present, the tension of the alveolar air and the blood alkalinity are within normal limits. The dyspnoea, when present, is due chiefly to **venous engorgement**, the result of myocardial insufficiency. This venous stasis affecting the pulmonary circulation produces deficient oxygenation, which finally results in cyanosis. Cyanosis without dyspnoea is most often found in chronic, decompensated mitral lesions. When the patients are resting quietly, dyspnoea is scarcely evident.

Visceral congestion varies considerably in the different valvular lesions. Congestion of the bronchial system is shown, at first, by symptoms and physical signs of bronchitis; in the more advanced cases, by pulmonary congestion and hypostatic pneumonia. Pleural transudates, especially right-sided hydrothorax, may be present; this preference for the right side has as yet not been satisfactorily explained. The liver may be slightly or tremendously enlarged and may even reach the pelvic brim. The enlargement may remain permanently. The shape of the liver is globular, its surface smooth. With gradual congestion of this organ, there is little or no tenderness on pressure; with sudden decompensation and consequent rapid engorgement, it is tender. The usual sites of tenderness are the epigastrium and the gall-bladder region. At necropsy the **spleen** in chronic valvular disease is often found enlarged and congested. This enlargement, however, often escapes diagnosis during life, for the edge of the organ is rarely palpable, and evidence derived from percussion alone is untrustworthy.

Renal involvement ranges from moderate passive congestion to sclerotic changes in the glomeruli. The former is common; the latter rare. Embolic renal infarcts are also rare in chronic rheumatic endocarditis. Renal congestion is shown by decrease in the amount of urine, by the presence of albumen and casts, and, in severe cases, by lessened excretion of phenolsulphophthalein (*q.v.*). Edema varies from the mild form found only at the ankles to general anasarca. Naturally, the latter is usually observed in extreme cases of long-continued decompensation.

Precordial pain in chronic valvular disease apparently depends chiefly upon the degree and suddenness of decompensation. Its distribution and character vary from a slight sensation of precordial heaviness

to agonizing attacks radiating from the chest to the left shoulder, left scapula, neck, abdomen, and, rarely, to the legs (Chapter XXI). Sudden dyspnoea and pain may occur as the result of a profound change in intracardiac circulation from embolus or infarct of the coronary or its branches.

The **arrhythmias** will be discussed in connection with the special features of decompensation found in the various valvular lesions.

SPECIAL SIGNS AND SYMPTOMS OF DECOMPENSATION IN CHRONIC RHEUMATIC VALVULAR LESIONS

Decompensated Mitral Regurgitation. — Venous stasis affecting the pulmonary circulation is an early symptom; hence bronchitis is one of the first manifestations of heart failure. Bronchitis may range from a short, hacking, unproductive cough to one with numerous soft, mucous râles and areas of sibilant breathing scattered over both lungs. The latter complex accurately resembles bronchial asthma. Sometimes bronchitis forms the predominant early feature of decompensation. Hemoptyses or expectoration of blood mixed with muco-pus are not infrequent. The patients are as a rule cyanotic. When cyanosis is extreme, the lips and extremities are bluish, the conjunctivæ suffused and discolored. Even with marked cyanosis, dyspnoea may be mild when the patient is resting quietly. Severe decompensation is accompanied by the visceral congestion already described. The usual subjective phenomena are a feeling of weight or pressure upon the chest; severe precordial pains are rare. On the other hand, sensitiveness to pressure in the epigastrium is common. Somnolence in the extremely decompensated cases with a fair output of urine, is probably due to cerebral congestion and edema.

Moderate pulse acceleration is the rule, extreme pulse acceleration or paroxysmal tachycardia is exceptional. The usual cardiac irregularities are extrasystoles (commonly ventricular) and auricular fibrillation; the extrasystoles are observed in the beginning; auricular fibrillation, in the later stages of decompensation.

Decompensated Mitral Stenosis. — A subjective feeling of palpitation combined with slight tachycardia is one of the earliest symptoms. It is due not only to moderate pulse acceleration, but also to increased violence of the heart action. Slight dyspnoea accompanies the palpitation. Cyanosis is a comparatively late symptom and marks the period of severe decompensation. An enlarged and distinctly pulsating liver may be present even when cardiac failure is not extreme. Sharp, precordial "sticking" pains are common; they usually accompany the tachycardia. Paralysis of the vocal cord due to paralysis of the left recurrent laryngeal nerve occasionally occurs.

Ortner in 1897 first described the connection between mitral stenosis and paralysis of the left recurrent nerve. Since then, reports of thirty-two cases

have been collected from the literature by Cuisset up to 1912. The various theories held accountable for the paralysis may be recapitulated as follows:

1. Ortnier's Theory: Recurrent paralysis is due to pressure of the enlarged left auricle upon the nerve.

2. Kraus' Theory: The right ventricular dilatation present in mitral stenosis causes displacement of the heart to the right, with consequent dragging on the aortic ligament and arch, and resultant stretching and paralysis of the nerve.

3. Alexander's Theory: The pulmonary artery, either by its own enlargement or indirectly by enlargement of the left auricle, is pressed against the nerve and aortic arch.

4. The nerve may be compressed and caught between bands of pericardial and mediastinal adhesions.

All these theories have had some necropsy support. Normally, the pulmonary artery is situated under, and divides immediately beneath, the aortic arch; below the pulmonary artery is the left auricle. It therefore seems improbable that the left auricle, even when extremely enlarged, can exert sufficient direct pressure to produce recurrent paralysis, unless the auricle is jammed between, or is adherent to, the pulmonary artery and aorta. This was the finding in one case that came to necropsy. In another case, the left auricle was the size of a small fist and was found pressing against the nerve. From careful anatomic studies of frozen sections, Fetterolf and Norris state that the effect of left auricular dilatation is pressure of the left pulmonary artery against the aorta, and of the left pulmonary vein against the pulmonary artery, thus forcing the latter against the aorta. They believe that direct pressure alone of the enlarged left auricle can scarcely produce paralysis unless the auricle is squeezed between or is adherent to the pulmonary artery and aorta. They conclude that anything which will dilate or force upward the left auricle, the left pulmonary vein, or left pulmonary artery will tend to produce paralysis, and that the latter must finally be caused by the nerve being "squeezed between the left pulmonary artery and aorta or aortic ligament." This conclusion bears out the common knowledge of the normal close juxtaposition of pulmonary artery and aorta. A similar conclusion was reached by Frischauer, who, in a case at necropsy, found the nerve compressed between the left pulmonary artery and aorta by the pressure of the dilated left auricle and pulmonary vein.

I had an opportunity of observing a patient with mitral stenosis in whom there was not only paralysis of the left recurrent nerve, but also a marked difference in the radial pulses. The history and the result of the examination in this case was as follows:

Female, aged 25, married two years, with no children or pregnancies, had a severe attack of inflammatory rheumatism thirteen years ago. There were no cardiac symptoms until three years ago; at that time palpitation began. During the last year, she has also become somewhat dyspnoeic. Hoarseness commenced six months ago. Examination of the larynx showed that, owing to left recurrent paralysis, the left vocal cord was immovable and somewhat shortened, and in the 'cadaveric' position. The voice was indistinct and hoarse. The examination of the cardiovascular system revealed the following: There was a noticeable difference between the right and left radial arteries on palpation, particularly when the arms were extended above the head. The systolic and diastolic blood pressures of the right brachial were respectively 110 and 80 mm. of mercury; of the left, 82 and 70 mm. There was no perceptible difference in the carotid pulsations. The pulse was regular, the rate 100 per minute. There was vigorous, visible, precordial pulsation; a diastolic thrill was felt, and a loud, rumbling, presystolic murmur was heard, at the apex. The aortic sounds were normal. Over the pulmonary area there was a somewhat rough systolic murmur and an exceedingly accentuated snappy second sound; the sharp click of pulmonary valve closure was also evident on palpation. A dry pericardial friction rub was heard over the lower sternum; three weeks later, there was evidence of fresh pericarditis over the pulmonary area. There was no systolic apical retraction. Orthodiascopic examination (Fig. 247) revealed a short aortal bulge (*A*); the outline of the aorta was apparently encroached upon by the very much dilated pulmonary artery (*P.A.*). The left auricle (*L.A.*), though not enlarged in the tracing, overlapped the pulmonary artery to some extent. The left ventricle (*L.V.*) and the right side of the heart

were also somewhat dilated. The electrocardiogram showed a negative *R III*, and a notched and enlarged *P* in the first lead.

In this case, there was no evidence of abnormal right ventricular enlargement; the heart was dilated to the left, as revealed by orthodiascopy.

The electrocardiogram (negative *R III*) added some corroboration. This evidence of left ventricular enlargement tends to discredit Kraus' theory as to the etiology of recurrent paralysis in mitral stenosis. The orthodiascopic findings and physical signs over the pulmonary artery in my case leads to the conclusion that the recurrent nerve paralysis was due to dilatation and pressure of the pulmonary artery against the aorta; whether or not the left auricle was a factor in pushing up the pulmonary artery it is impossible to state. Lian and Marcocelles insist on the presence of old thrombosis of the left auricle, or of a mediastinitis as contributory causes. Pericarditis was present in my patient; it was at first confined to the precordial area over the lower sternum; later, it involved the area over the pulmonary artery. This may have been an added factor in binding down the dilated pulmonary artery.

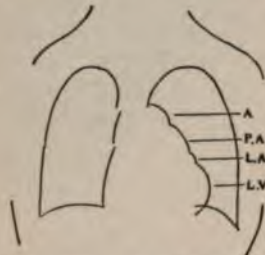


FIG. 247. — Orthodiascopic tracing: *A*, aortic outline; *P.A.*, outline of pulmonary artery; *L.A.*, outline of left auricle; *L.V.*, outline of left ventricle.

A unique feature was the presence of a marked difference in the right and left brachial blood pressures and in the radial arteries; this, in conjunction with paralysis of the vocal cord, formed a striking resemblance to the clinical syndrome of aortic aneurism. The difference in brachial pulse pressure seemed due to the dilated pulmonary artery being wedged under the aortic arch opposite the origin of the left subclavian artery, with a pressure sufficient to produce interference with the circulation of the subclavian, and consequently of the left brachial.

Attacks of paroxysmal tachycardia are frequent in decompensated mitral stenosis. Emboli produce occasional complications. Hemiplegia and aphasia may follow cerebral emboli. As an instance, one of my patients, a woman aged 50 with a mitral stenotic lesion, had aphasia for 10 years from a cerebral embolus. A large artery of the extremity may be plugged by an embolus and cause gangrene. This happened in a patient of 40 with mitral stenosis and finally led to amputation of the leg; the patient recovered.

Severe decompensation in mitral stenosis is usually brought about by the development of **auricular fibrillation**, with the usual signs and symptoms already described. The arrhythmia, once established, usually becomes permanent. Occasionally, however, fibrillation occurs with each fresh exacerbation of endocarditis. Auricular fibrillation is found oftener in mitral stenosis than in any other valvular disease.

Aortic Stenosis. — The symptoms of heart failure depend chiefly upon the degree of ventricular hypertrophy with its implied lowered cardiac efficiency. Marked decompensation is a late symptom, and is found only when hypertrophy becomes extreme. Congestion and auricular fibrillation occur only in the terminal stages. Among earlier symptoms are those due to cerebral anemia (dizziness and faintness). Other beginning manifestations consist of a subjective feeling of palpitation and of moderate tachycardia. Embolic infarcts in the viscera or extremities are occasional complications.

Aortic Regurgitation. — The patients are characteristically prone to suffer from attacks of tachycardia (often paroxysmal), and from very severe precordial pains. Pains and tachycardia often occur in nocturnal attacks. The pains are usually severe, sometimes agonizing. Their favorite site is the precordium; they may radiate to the arms, neck, abdomen, and even to the legs. Fright, excitement, over-exertion, or nervous strain may be sufficient to initiate an attack of pain or of tachycardia, especially when ventricular hypertrophy is far advanced. Such attacks may also represent the only evidence of fresh endocarditis. Susceptibility of the heart to nerve influences of the most varied kinds, a condition so common in aortic regurgitation, depends chiefly, I believe, upon the continued mechanical insults from aortic hyperactivity to the branches of the cardiac plexus surrounding the root of the aorta. This may well produce an altered state of nerve tone and of nerve control, leaving the heart readily susceptible to extraneous influences.

Hemoptyses occasionally occur with the tachycardial attacks; they seem due to acute pulmonary congestion from rapid heart action. Hemoptysis may also be the result of a pulmonary infarct. Another occasional complication of aortic regurgitation consists in periods of **somnolence** merging into stupor. In one of my patients, a girl of 17 with a typical rheumatic regurgitant lesion and tremendous left ventricular hypertrophy, these cerebral manifestations were usually ushered in by fever up to 103°, and by paroxysms of auricular fibrillation. Cyanosis was not present. The dyspnoea during these attacks was not more marked than usual. The fever and fibrillation seemed due to recrudescence of endocarditis. Meningitis could be excluded. The accompanying somnolence, and later, stupor, in this and other attacks, could scarcely be accounted for by the fever alone or upon the supposition of a toxemia, for the patient had had a severe and long-continued pneumonia with higher fever, without any cerebral manifestations. Perhaps cerebral anemia from disturbed circulation in the brain is the underlying cause for attacks of somnolence and semistupor in patients with well-marked aortic regurgitation.

Visceral congestion, edema, and permanent auricular fibrillation represent late stages of decompensation in aortic regurgitation.

THERAPY OF RHEUMATIC ENDOCARDITIS

In **acute rheumatic endocarditis**, or in endocarditic exacerbations accompanied by general rheumatic manifestations, the salicylates, preferably salicylate of soda, are indicated. Clinical observation has shown that the salicylates often control general rheumatic manifestations; it is therefore assumed that they may control the rheumatic poison affecting the heart. I prefer to give the salicylate of soda in 15 grain doses hourly until tinnitus occurs, or until six doses have been

taken; thereafter, the same dose should be given three times daily. The futility of sera and vaccines is discussed later (Chapter XVI). Rest in bed should be enjoined during the period of the acute endocarditic attack. The plan, sometimes followed, of keeping patients in bed many weeks, or even months, after the acute stage has passed, possesses no value in aiding the circulation or in preventing reinfection (Chapter XVIII). From three to five weeks I consider the average time for rest in bed after the acute manifestations have passed. In those with rapid heart action or with precordial discomfort, an ice bag should be placed over the heart; it should be kept in a proper sling so that the patient is not continually annoyed by its slipping. The length of time that ice is to be applied should be governed to a great extent by the patient's feelings. I have occasionally found more relief from hot applications (hot water bag) than from cold. If tachycardia is marked, or if patients are restive, the bromides alone, or combined with small doses of morphine, are indicated. Occasionally, hypnotics (chloral, veronal) are required. Digitalis is not indicated, for decompensation does not occur in acute endocarditis. An exception may be the presence of tachycardia with acute dilatation and dyspnoea, a condition more frequent in children than in adults.

In **chronic rheumatic endocarditis**, when no decompensation exists, drug medication intended to affect the heart and circulation is not indicated. Here, questions respecting proper exercise, vocation, and occupation play important rôles (Chapter XVIII). The therapeutics of **decompensation** in chronic endocarditis is described, to a great extent, in another chapter (Chapter XVI). Though medication is, in general, the same for the various valvular lesions, it should again be indicated that the same therapy is not followed by uniform results. The most beneficial effects are derived from digitalis when employed in decompensated mitral stenotic lesions with auricular fibrillation; this follows from the almost specific action of digitalis (*q.v.*) upon the conduction system. In the heart failure of aortic disease with marked hypertrophy digitalis has little or no effect in relieving decompensation, even in the presence of auricular fibrillation.

PROGNOSIS

For clinical purposes, the question of prognosis in rheumatic endocarditis may be divided into the **acute and subacute**, the **quiescent** and the **chronic** stages. The first two are here grouped together because they overlap so frequently that a sharp distinction is impossible.

In the **acute and subacute** stages, the main prognostic problem is to determine, if possible, the virulence of the infection. Although, as stated, a few observers claim to have isolated the specific organism from the blood of patients suffering from rheumatic endocarditis, this has

not been substantiated by other careful bacteriologists; hence, the blood examination for bacteria cannot at present be used to gauge the severity of the infection. In addition to information gained by physical signs, most reliance regarding the degree of virulence in rheumatic valvular infections must be placed upon such clinical manifestations as the acuteness and severity of onset, the frequency of chills, the presence of hyperpyrexia, vomiting, leucocytosis, severe anemia, delirium, and upon such complications as pericarditis and pleurisy with effusion. If both the aortic and mitral valves are affected, the acute and subacute stages are apt to last a longer time than when one valve alone is diseased, and the prognosis correspondingly worse. Patients with aortic lesions are somewhat more prone to complications arising from the virulence of the infection than are those with mitral lesions. During the time that rheumatic manifestations are present, the endocarditis cannot be regarded as quiescent, even though there be no clinical manifestations of recrudescence. However, more direct evidence of fresh endocarditis may be found in increased intensity and harshness of endocardial murmurs, temporary arrhythmias, precordial "sticking" pains, and, occasionally, in embolic infarcts in the brain, lungs, or kidneys. Slight rises of temperature, if not otherwise accounted for, are to be regarded as probable signs of the continuation of the endocarditic process. Another highly suggestive evidence is anemia, severe or moderate, not responding to the usual treatment.

Occasionally the infection is mild, fever and other inflammatory manifestations cease, the valvular lesion quickly becomes quiescent, and indeed soon eludes all signs of ever having been present. Such cases usually occur in children and young adults, and apparently represent instances in which healing begins soon after the mild endocardial infection has run its course. The existence of such cases has been corroborated by necropsy findings.

If the physical signs or clinical phenomena indicate an active endocarditis lasting for a month or longer, the outlook for a "quiescent" period, and therefore for a favorable prognosis, becomes correspondingly poor. These patients may die of embolic infarcts, or of superadded bacteremias, such as pneumococcemia or streptococcus viridans infections. Decompensation usually plays a minor rôle.

Quiescent Stage.—In the fortunate cases, this stage represents uninterrupted convalescence from the acute and subacute, with complete cessation of active signs of endocarditis. These patients with their recurrences and exacerbations comprise the great class of chronic rheumatic heart cases. Regarding the quiescent stage as convalescence from the acute, decompensation is rare, the chances of the lesion becoming 'chronic' and remaining quiescent are good. The prognosis then depends upon that of chronic endocarditis.

In **chronic quiescent** cases, the cardinal point in prognosis is the consideration, not of the number or varieties of murmurs present,

but the extent and type of damage to the myocardium, factors usually decided by a study of the muscular and circulatory efficiency of the heart. If myocardial efficiency and cardiac reserve are good, a correspondingly good prognosis may be given. Regarding prognosis from the standpoint of longevity, the above observations must be combined with a general knowledge of the average duration of life in the various types of valvular lesions; but each case must nevertheless be studied individually in the attempt to gauge the probable span of life.

It has been demonstrated statistically that patients with mitral insufficiency live longest; individuals with this lesion living to ripe old age are not uncommonly encountered. The usual duration of life in mitral stenosis is much less; one of the reasons for this is that such cases are prone to develop auricular fibrillation, and with it, the usual train of symptoms and dangers arising from decompensation. In aortic regurgitation, massive ventricular hypertrophies (*cor bovinum*) are common; yet years may elapse until death occurs from decompensation, emboli, or from some superadded acute infective process. In general, however, the chances for longevity with this lesion correspond somewhat to the degree of ventricular hypertrophy; where this is marked or extreme, patients are not apt to live beyond middle age.

ACUTE BACTERIAL ENDOCARDITIS

Other terms for this affection are malignant, ulcerative, infectious, and septic endocarditis. Bacterial endocarditis as an acute process engrafted upon a chronic valvular lesion is not here included.

It has already been pointed out that, although not proven, rheumatic endocarditis is probably of bacterial origin. But acute bacterial endocarditis differs so markedly from the rheumatic type, and even from streptococcus viridans infection (itself a bacteremia), that it deserves individual description.

Acute bacterial endocarditis is almost always a secondary process; its presence as a primary lesion has indeed been denied by several writers. It may complicate erysipelas, pneumonia, puerperal fever; in fact, any bacteremia. It is occasionally present as a terminal infection. Although the course of acute bacterial endocarditis is essentially acute, occasional cases of comparatively long duration have been described. In acute staphylococcic infections, the portal of entry is the skin or an osteomyelitic focus; in streptococcus pyogenes, the mucous membrane of the throat or uterus. Gonococci, and also streptococci and staphylococci, may enter the general circulation from the genito-urinary tract; pneumococci, from the respiratory tract.

The **clinical picture** of acute bacterial endocarditis is essentially that of a more or less virulent bacteremia, with all its protean and manifold characteristics. The disease rarely lasts more than four

weeks. There are often no cardiac manifestations; no murmurs, pain, dyspnoea, or other signs of cardiac involvement.

The clinical manifestations are sometimes divided into the typhoid, septicæmic, cerebral, etc. These varying characteristics depend upon the virulence of the invading organism, and upon the underlying cause of the bacteremia (erysipelas, puerperal septicæmia, etc.).

The **course** of acute bacterial endocarditis is usually marked by high fever; daily chills are common; long remissions of temperature are rare. Sweating is often profuse. The onset of the endocarditis may be marked only by an intensification of the fever from the original source of infection, or by an initial rigor. Added hints of its onset may be found in sudden increased pulse rapidity, in sticking pains in the pre-cordium, in the friction rub of dry pericarditis. When the latter is present, an endocardial involvement as well is usually present.

Septic infarcts into various organs mark the stormy progress of the disease. Thus, cerebral infarcts may produce paralysis and unconsciousness, or, occasionally, purulent meningitis; symptoms from the latter may then dominate the clinical picture. Hematuria, sometimes abundant, may follow a renal infarct; pneumonia, an infarct in the lungs. Of frequent occurrence are **petechiæ**. They are usually numerous, especially upon the oral and conjunctival mucous membranes. The petechiæ sometimes contain pin-point pustules in the center. Occasionally, patchy erythematous areas in the skin, resembling urticaria, may be found. Various joints, especially the larger ones, may be involved in a purulent or sero-purulent inflammatory process.

The usual general manifestations of sepsis, or of a septic 'typhoid' condition, are common. The patients may be actively delirious, or be stuporous and comatose. There may be *coma vigil* with a dry and coated tongue.

Medication, including sera and vaccines, possess no value. The treatment is symptomatic only.

SUBACUTE AND CHRONIC STREPTOCOCCUS VIRIDANS INFECTIONS

I have made no attempt to divide the disease into two distinct stages, for the terms "subacute" and "chronic" depend chiefly upon differences of duration rather than of symptomatology. The usual duration is from a month to one year or more, hence either term may be appropriate to the individual case.

Pathological Features. — Of the chronic bacterial infections of the heart, that by the streptococcus viridans has been most exhaustively studied. The process is almost always engrafted upon a chronic rheumatic infection. Occasionally, congenital lesions form the nidus. The pathological process consists of vegetative proliferative masses of grayish, greenish, or pink color. Their main site is the mitral valve. Here they may form a few soft, friable masses, or the valve may be encrusted

with large polypoid lesions. The latter may then extend along the left auricular wall above, and the chordæ tendinæ below. The process on the chordæ tendinæ occasionally leads to ulceration and rupture of these structures. Similar sequelæ are sometimes found as the result of vegetations on the mitral. Proliferative lesions on the aortic cusps and walls are less common and less extensive than upon the mitral. Mycotic aneurisms of the valves are occasionally found.

Characteristic changes in the **kidney** depend chiefly upon the presence of infarcts. When pyogenic, they give rise to numerous small congested areas containing minute purulent foci; these are readily seen when the capsule is stripped from the kidney. Non-pyogenic bland infarcts occasionally occur; they show the changes usual to anemic necrosis; the infarcted areas are wedge-shaped and may be several centimeters in depth. When they are recent, the cut surface is yellow; the color becomes paler with the process of organization. Another type of infarct, sometimes assumed as pathognomonic of chronic streptococcus viridans, is embolic focal nephritis. Characteristic changes are then found in the glomeruli; a part of, or an entire tuft, may be involved. The capillaries are congested, the glomeruli contain a fibrinous exudate, the glomerular epithelium becomes swollen, and finally desquamates. The adjacent parietal layers of Bowman's capsule are often involved in the necrotic process, so that the entire necrotic area becomes semilunar in shape. The mass eventually organizes. There is a growth of epithelial cells from the healthy adjacent Bowman's capsule, finally covering the lateral surface of the mass. When healing is completed, the result is a hyaline area of pyramidal shape.

The **cerebral lesions** consist chiefly of areas of softening, following emboli. Occasionally, cerebral hemorrhages from rupture of embolic aneurisms of the cerebral vessels have been observed.

Clinical Features. — The pathological features enumerated, and the fact that streptococcus viridans infection is a bacteremia, make it apparent that the clinical complex varies considerably. The predominant clinical features depend upon the toxemia, upon focal embolic insults in the various organs and viscera, and upon the pronounced pathological changes in the heart and kidneys.

The **onset** of the streptococcus viridans affection is usually insidious; the only complaint for days or weeks preceding the definite febrile period may be slight general malaise or mild anorexia. Sometimes the initial symptoms resemble those of other febrile invasions: rigors, chills, vomiting, and fever between 101° and 103°. Occasionally the onset is even more acute, and the daily temperature reaches 104° or 105°, with remissions resembling malaria. Such initial symptoms usually betoken a stormy and comparatively short course for the viridans infection. When the disease is once established, it is usual to have a febrile period from one to three weeks, with afebrile intervals of about the same duration. Each new invasion, at its beginning, may be

marked by severe chills and hyperpyrexia; temperatures up to 106° are occasionally encountered. The disease may thus continue for months until emboli or exhaustion produce death.

Petechiæ represent one of the most prominent and frequent manifestations. They are probably due to small emboli. On the mucous membrane, they may appear as small, isolated red spots, with pale centers surrounded by an area of congestion, and sharply defined from the normal mucous membrane. The favorite site of petechiæ is the lower conjunctiva. Here they are characteristically found at the angulation of the vessels; the entire sac should be well exposed by pressing back the eyeball. Occasionally, petechiæ are found, not in the lower, but in the upper lid. They are also found upon various parts of the oral and even pharyngeal mucous membranes. In one of my patients, in whom petechiæ often appeared in crops on the skin as well as on the conjunctivæ and buccal mucous membrane, I found, in addition, a few in the anterior urethra.

Skin petechiæ resemble those on the mucous membrane. They are usually small and punctate; occasionally, they become somewhat larger. Very rarely, they are surmounted by minute pustules. While isolated petechiæ are the rule, it is not unusual to have them appear in crops over various parts of the body. Occasionally their appearance is attended by severe neuralgic pains in the area corresponding to the location of the rash. The cause of such pains is not apparent. The petechiæ are not tender; they usually disappear within one week. They sometimes leave small pigmented spots for a few days, which must be carefully distinguished from other pigmentations, especially freckles.

In addition to petechiæ, **other skin manifestations** are not uncommon in this infection. Painful erythematous nodules, slightly red and somewhat tender (Heubner's nodes), may occur in the terminal phalanges, especially of the fingers. The nodules, usually multiple, disappear within a few days; they are probably of embolic origin. Pyogenic emboli in the deeper dermal arteries may produce skin gangrene or necrosis. This occurred in one of my cases and finally resulted in sloughing of the entire scrotum. Emboli are sometimes scattered shower-like through the smaller arteries of the extremities, producing numerous small areas of deep or superficial gangrene, and necrosis in fingers and toes. This was well illustrated in a woman aged 50 with streptococcus viridans and a decompensated double mitral lesion. She developed gangrenous areas on several digits and toes, and on the dorsum of both hands. She died one week later.

The **hematological examination** is of great importance. While a mild secondary anemia with moderate leucocytosis and a high polynuclear count is the rule, anemia may become so marked as to overshadow the clinical picture, even leading to the erroneous diagnosis of non-organic, instead of organic, endocardial murmurs. Occasionally,

the blood picture and the hemoglobin percentage so closely resemble pernicious anemia that patients have been treated for the latter disease; only at necropsy was the correct diagnosis established.

Splenic enlargement is quite common; it may vary from moderate increase in size to one reaching several inches below the free border of the ribs. At necropsy, infarcts of various sizes are almost regularly found in this organ.

Changes in the Urine.—A trace of albumen and a few casts are frequently found. More significant, when traced to the kidney as its source, is the presence of a microscopic amount of blood. Rarely, macroscopic hematuria is present. The streptococcus viridans can only occasionally be cultured from a catheterized urine specimen. In exceptional instances renal signs and symptoms predominate in the clinical picture. This occurred in one of my patients. There was unilateral renal colic and bleeding. A blood culture taken at that time was sterile. A nephrectomy was done; the affected kidney was found twice the normal size and riddled with infarcts. The patient died two weeks later. Another blood culture, taken two days before death, was positive. At necropsy, valvular lesions characteristic of streptococcus viridans were found.

Nervous manifestations are frequent. They depend upon one or several factors, all of which must be etiologically considered. Stupor, for example, may be due to toxemia, hyperpyrexia, meningitis, cerebral infarcts, or to uremia. An instance of the difficulties with which the etiology of this one symptom may be surrounded is that of a woman aged 45, admitted to the hospital in semi-stupor. A double mitral lesion, auricular fibrillation, and irregular fever were present. The urine showed microscopic hematuria and a few renal elements. Two blood cultures were negative. A spinal tap was unsatisfactory. Semi-stupor finally merged into coma and death. Clinically, the symptoms seemed due to meningitis. At necropsy, a double mitral lesion, splenic infarct and the glomerular kidney changes of streptococcus viridans were found. Meningitis was not present. Toxemia was the apparent cause of stupor and coma. The most common cerebral lesions in streptococcus viridans infections are hemiplegias; monoplegias are rarer. Occasionally, pareses, not paralyzes, occur, to disappear during the course of the disease. Meningitis is usually a terminal event; its clinical signs may be disguised by toxic symptoms.

Pneumonic complications are not often found in chronic streptococcic viridans infections. **Enterorrhagia** from an infarct is occasionally met with. **Retinal hemorrhages** sometimes occur.

Cardiac symptoms (precordial discomfort, decompensation, etc.) are not regularly present, and then only late in the infection. Even when valvular vegetations are extremely exuberant, decompensation may not occur during the entire course of the disease. On the other hand, physical signs of a valvular lesion are usually frank and unmistakable.

This is because of a preëxisting valvular lesion, and because the streptococcus viridans usually produces abundant valvular vegetations. Occasionally, when the mural instead of the valvular endocardium is chiefly involved, murmurs may be faint and not characteristic. Arrhythmias, especially auricular fibrillation and extrasystoles, are not uncommon. When found during the active febrile periods, they are apparently caused by endocardial, rather than by myocardial, involvement, for they usually cease with the disappearance of fever. When present during decompensation, cardiac irregularities are probably due to myocardial degeneration, or are indicative of myocardial insufficiency.

The study of **blood cultures** is naturally of extreme diagnostic importance. When properly made and a sufficient length of time (one week) is allowed for the appearance of a growth, the organism has been recovered in a great majority of the cases. Sometimes numerous blood cultures are necessary before the organism can be isolated.

The clinical course of some cases in which the coccus has not been recovered during life, and the presence at necropsy of glomerular lesions characteristic of streptococcic viridans infections, make it probable that some cases heal, or at least remain quiescent over a long period of time. The **quiescent interval** may last several years. These have been grouped as being in the "bacteria free stage" (Libman). The following cases are illustrative of this condition:

Male, aged 21, had scarlet fever, followed by severe endocarditis when 7 years old. A double aortic and mitral regurgitant lesion resulted. For many years there was no decompensation or other symptom except occasional attacks of hematuria. A few months before death the patient developed chills and fever; the streptococcus viridans was then found in the blood. In the light of our present knowledge this case seems to belong to the category just described. The intervals between the attacks of hematuria probably represent long remissions or "bacteria-free stages."

Male, aged 40, entered the hospital with fever. He had a mitral and an aortic lesion. Blood cultures on two occasions showed the streptococcus viridans. The fever remained high for three weeks, during which time the patient was delirious. The temperature then gradually became normal. The patient was not seen again for one year. In this interval he said that he had been quite well. He was readmitted to the hospital with a pneumonia of embolic origin. The physical signs of valvular disease were the same as upon first admission. Blood cultures were negative. The patient made a good recovery from the pneumonia.

The **extent and frequency of bacterial reinvasions** (*i.e.* the duration of the bacteria-free interval) are at least partly governed by certain characteristics of the vegetations. If the latter are covered by a firm, partially organized fibrin layer, the bacteria are not as apt to invade the blood stream; while if the fibrin layer is thin or is so situated that

the blood stream impinges with full force against it, the bacteria are much more likely to be washed into the blood current. The amount of fibrin covering the vegetations probably depends upon the virulence and proteolytic digestive power of the invading organism.

Besides symptomatic treatment, **therapy** consists in the use of vaccines, preferably autogenous, and of horse serum sensitized to various strains of the streptococcus viridans. An occasional cure by these methods has been reported. In view of the exceptionally long remissions, sometimes for years without the use of vaccines, final judgment regarding "cures" by serum or vaccine treatment should for the present be withheld. In my own cases I have observed no effect upon the course of the disease from serum or vaccine therapy; nor was there any evident arrest of the pathological process. This was shown by the examination of such hearts at necropsy. For example, in one case in whom the diagnosis of streptococcus viridans infection was established by blood culture four days after the initial chill, not only did autogenous vaccines and sensitized horse serum administered for months have no effect upon the symptoms or course of the disease, but the post-mortem examination showed exuberant and fresh masses of vegetation literally plastering the mitral valves, the left ventricle, and auricle. Unfortunately, therapy of all kinds, including intravenous injections of silver salts (Chapter XVI), must at this time be regarded either as futile or as purely in the experimental stage. The wisdom of prophylactic vaccination against viridans infection in those cases in whom its later occurrence is feared deserves careful consideration.

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CHAPTER XIV

CARDIAC SYPHILIS

As a rare complication in the **secondary** stage of syphilis, it has been found, pathologically, that the myocardium alone may be occasionally involved. Such involvements may be sufficient to produce symptoms of myocardial insufficiency: dyspnoea on exertion, indefinite precordial distress and pain, precordial sensitiveness to pressure, slight pretibial edema, sometimes also a faint systolic murmur at the apex.

The symptoms of cases in the **tertiary** stage depend upon the degree, extent, and admixture of muscular, valvular, and endocardial involvement.

In occasional cases, isolated luetic valvulitis of the **mitral** or **tricuspid** has been found. In a vast majority of instances, however, the **aortic valves** bear the brunt of the endocardial infection. In addition to the cusps, there is often widespread destruction of the aortic walls, finally resulting in **aneurismal dilatations** (*q.v.*) and in **true aneurisms**. Accompanying the aortitis are changes affecting the myocardium, the remainder of the endocardium, and the coronaries. Together, they comprise a composite picture often similar to that found in non-luetic cardiosclerosis (Chapter XII).

In the absence of a history of venereal infection or of other signs of syphilis, the presence of a strongly positive Wassermann blood reaction is of paramount importance in the diagnosis of cardiovascular syphilis. More than one blood examination may be necessary, or provocative injections of mercury, or small doses of salvarsan may be required in order to obtain a positive Wassermann reaction. If the blood Wassermann remains negative, the chemical and cytological examination of the spinal fluid may indicate the presence of syphilis. Thus tested by routine and other methods, the diagnosis of cardiovascular syphilis can be substantiated in the great majority of suspected cases.

From the **clinical** standpoint, the characteristic symptomatology and signs of **tertiary cardiac syphilis** depend mainly upon the presence of **aortitis** alone, or upon it as part of the picture of cardiosclerosis. In

the exceptional instances in which the myocardium alone is affected, the diagnosis rests upon the usual manifestations of severe myocarditis. The possibility of gummata in the myocardium must also be considered. They add to the probability of the occurrence of a myomalaceous focus, or, when involving the conduction system, upon the presence of heart block.

ANEURISMAL DILATATIONS OF THE AORTA

A century ago Hodgson made the fundamental distinction between sacculated and "dilatation aneurisms." He defined the latter as a "preternatural permanent enlargement of the cavity of an artery." The former is a definite protrusion of only one side of its wall and may include a small or large part of the arterial caliber. This distinction is of clinical importance in aortic disease. The French still apply the term "*Maladie de Hodgson*" to aneurismal dilatations of the aorta associated with valvular insufficiency. Aortitis of the ascending and transverse aorta is now known to be of luetic origin in the great majority of instances. The pathology and mechanism of aneurismal dilatations of these parts of the aorta have been described in detail by Thoma, who had studied ninety-two cases. He ascribes their fundamental cause to lessened resistance of the arterial walls, and categorically states that "such weakened condition is not found in the descending thoracic aorta," which showed no change macroscopically. The pathological process in dilatations of the first and second parts of the aorta consists of a mesaortitis, with perivascular infiltrations of the vasa-vasorum, small-celled or granulomatous infiltration in areas of the media, and splitting and destruction of the muscular and elastic tissue layers. Diffuse dilatation of the entire thoracic aorta from its root to the diaphragm is occasionally seen. I have observed three such cases: one was an autopsy finding and included severe myocarditis and coronary sclerosis, the other two were diagnosed by physical signs in conjunction with fluoroscopy.

The **orthodiascopic picture** of dilatation of the **first portion and arch** of the aorta has already been described (Chapter IX). On auscultation the normal first sound at the right base is accompanied or replaced by a soft blowing murmur, usually not transmitted beyond the first and second right interspaces. Occasionally the first sound is faint or absent. The second sound is accentuated, of varying quality, and is best described as bell-like, metallic, or tinny; it is often followed by a soft diastolic murmur, indicative of aortic insufficiency. The accentuated and metallic quality of the second sound is not always due to extreme hypertension, for it is frequently present with normal or very slightly raised systolic blood pressure. The factor most responsible for this abnormal second sound is, I believe, the aortic dilatation acting acoustically as a sounding box in reflecting and thus changing the character of the sound.

In marked aneurismal dilatation, especially of the arch of the aorta, there is often visible aortic pulsation in the jugulum. When not visible, it may be felt by insinuating the finger behind the manubrium sterni. In cases of aneurismal dilatation of the first part of the aorta, there may be more marked right than left carotid pulsation, a fact determined by placing the finger deeply behind the respective clavicles. At the apex, there is frequently a soft systolic murmur, probably due to relative mitral insufficiency, or there may be a loud murmur due to thickening of the mitral cusps and mural endocardium. If the aneurismal dilatation is accompanied by left ventricular hypertrophy and by hypertension, there may also be a reduplicated apical impulse or a somewhat accentuated first sound at the apex, as well as a slightly metallic second sound.

Reports of aortitis are almost entirely confined to disease of the ascending or transverse aorta with fragmentary or no reference to the descending thoracic. McCrae, Allbutt, Osler, and others consider aneurismal dilatation of the descending thoracic aorta extremely infrequent, a statement apparently based upon post-mortem findings, for no symptomatology is mentioned nor had a clinical diagnosis been made. My object is to indicate through illustrative cases the comparative frequency, diagnosis, symptomatology, and therapy of aneurismal dilatations of the **descending thoracic aorta** and to present its claim to a clinical entity.

Male, age sixty-seven, had never been seriously ill previous to his present complaint. He had been a heavy smoker and had suffered from a venereal infection forty years ago, for which he had received many subcutaneous injections (presumably of mercury). His present illness began two years ago with exceedingly mild symptoms: very slight precordial pains when lying on the left side, and slight dyspnoea upon climbing stairs. Upon examination, the patient looked well preserved, the carotid pulsation on both sides was somewhat exaggerated, there was a belt of small dilated capillaries over the lower part of the chest. The cardiac thrust at the apex and the systolic impact at the right base were somewhat exaggerated. The cardiac area seemed normal to percussion. At the base there was a double murmur, a rough systolic and a softer diastolic, transmitted and best heard to the left of the middle third of the sternum; a definite sensation of heaving impulse was imparted to the examining hand placed over the same area. Similar but fainter murmurs were heard at the apex. Both radial pulses were alike: the average systolic blood pressure was 170 mm., the diastolic, 70. Neurological and other examination revealed no abnormalities. There was slight pretibial edema. The Wassermann reaction was negative. The roentgenogram (Fig. 233, Plate XIX) and orthodiascopic tracing (Fig. 232) showed a long fusiform dilatation of the entire descending thoracic aorta. The electrocardiogram presented evidence of left ventricular preponderance, *i.e.* negative *R III*. Treatment consisted

of one salvarsan and many mercurial injections; iodide of potash in moderate doses was administered in alternate periods of two weeks. Tincture of digitalis was given for one week until the edema of the legs disappeared. The patient has been under observation for several years; he has been quite comfortable, with no pains for more than one year; the physical signs along the middle third of the sternum have markedly receded.

Female, age forty-seven years, married sixteen years, had never been pregnant. Prior to the present illness she complained of dyspnoea on exertion, and of nocturnal palpitation accompanied by pain in the lower precordium. She lost about forty pounds since her illness began. One week before admission to the hospital her legs became edematous. The patient was emaciated, the right pupil was somewhat larger than the left, both reacted sluggishly to light but normally in accommodation; the knee reflexes were diminished; Romberg's sign was absent. The patient was dyspnoeic even when at rest. There was vigorous, visible carotid and jugular pulsation; the aortic thrust could be plainly felt by pressing the finger tip behind the manubrium. By placing the eye on a level with the patient's chest, two distinct areas of impact were discernible; one corresponding to the apical region, the other to an area slightly to the left of the lower half of the sternum. The latter impact was also palpable by sinking the fingers in the lower left intercostal spaces, and also by auscultating with the stethoscope over this area in the routine manner. There was no precordial tenderness. The apex was felt most distinctly in the sixth interspace, a considerable distance (14 cm.) from the midsternal line. Over the base, and particularly to the left of the midsternum, there was a rough systolic murmur and a somewhat accentuated second sound merging into a soft murmur occupying the entire diastole. Friction sounds indicative of dry pericarditis were present at the base and apex. There was slight edema of the legs. The Wassermann reaction was negative upon first examination; some months later it became positive. The systolic blood pressure ranged between 200 and 180 mm., the diastolic between 100 and 40 mm.; the pressure in both arteries was equal. The urine was normal. The roentgenogram (Fig. 234, Plate XX) showed fusiform aneurismal dilatation of the entire descending thoracic aorta; orthodiascopic examination corroborated this finding. The electrocardiogram presented evidence of left ventricular preponderance. The patient refused treatment; she has since reentered the hospital, with signs of severe cardiac failure. She subsequently received iodide of potash and mercurial injections, with marked improvement.

Male, age fifty-three years, laborer, married. Venereal infection denied. Three and one half years ago substernal pains and dyspnoea upon walking appeared. One year ago he was compelled to stop work because of the frequency and severity of these attacks. The pains usually radiated to both sides of the chest, the shoulders, and right side

of the head. During the last few months he had frequent pharyngeal spasms upon attempting to swallow fluids, so that there was occasional regurgitation through the nose. There were no gastric symptoms. The patient looked florid and well nourished; there was no dyspnoea when at rest. Upon admission to the hospital the right radial pulse was much smaller than the left; there were no abnormal carotid or jugular pulsations. The apex beat was best heard in the fifth interspace, 7 cm. from the midsternal line. On auscultation the first sound at the apex was scarcely audible; the second was normal. The first sound at the right base could not be heard; the second aortic was normal. Both were distinctly heard to the left of the middle third of the sternum, the first sound there being normal in intensity, the second somewhat accentuated. There was no edema of the legs. Examination of the other organs revealed nothing abnormal. The Wassermann test was negative. An interesting phenomenon was the fact that intermittently for several weeks the right radial was smaller than the left, the difference sometimes amounting to 40 mm. of mercury in the systolic brachial pressures. These inequalities had no relation to the symptoms. The roentgenogram showed a distinct, somewhat tubular shadow behind the hypertrophied left ventricle. With the orthodiascope the shadow of the dilated descending aorta could be faintly seen through the upper ventricular shadow. The electrocardiogram presented the usual evidence of left ventricular preponderance. The patient was given three injections of salvarsan intravenously, and numerous mercurial injections in combination with iodide of potash. The pains and pharyngeal spasms gradually subsided; the pulse inequalities became only occasionally noticeable and finally disappeared. The abnormal physical signs to the left of the sternum markedly receded. The patient returned to work; his present complaint consists in slight occasional substernal pain on walking.

Male, age thirty-eight years, a vigorous and healthy-looking man, complained during the last six months of slight dyspnoea upon climbing stairs, but none when at rest. He was a heavy cigar smoker. He had gonorrhea twenty years ago and denied any other illness. The blood pressure was normal. There was vigorous carotid pulsation at the root of the neck. There was no pain on precordial pressure. The apex beat was strong, and was felt most plainly in the fourth interspace, 11 cm. from the midsternal line. A soft systolic murmur was heard at the apex. At the base the first sound was impure, the second not accentuated but prolonged and somewhat liquid in character occupying the entire diastole. These abnormal sounds were heard loudest and most distinctly along the middle left sternal border. The eye placed on the level with the chest could discern a slight systolic heave over the latter area, apparently distinct from that at the apex. The other organs were normal. The Wassermann reaction was negative. The roentgenogram showed dilatation of the upper part of the thoracic aorta, the latter

being visible as a somewhat denser shadow behind the ventricles. The orthodiascope confirmed this. The electrocardiogram was normal. The patient was given salvarsan, .3 gm. intravenously, and many bichloride injections combined with iodide of potash. The dyspnoea has entirely disappeared; the abnormal sounds at the base and left sternum are much less pronounced than at the first examination.

Male, age fifty-three, entered the hospital in the surgical service of a colleague and did not come under the writer's observation. The history was that of an esophageal stricture situated in the mid-thoracic region. The aneurismal dilatation was discovered in making a routine roentgenographic examination of the chest. The stricture was presumed to be due to compression by the dilated aorta. The Wassermann test was not done. The patient passed from observation. The case is included here because the roentgenogram is typical of aneurismal dilatation of the descending aorta.

Symptomatology. — Sharp, continuous, gnawing pains, such as those often associated with sacculated aneurism, are not prominent features of dilatation of the descending aorta. When present, substernal pains or those referred to different parts of the chest, neck, or head are most apt to occur with exercise. It is difficult to state the exact etiology of these pains. They are probably not due to pressure of the dilated aorta upon the surrounding structures (esophagus, ribs, dorsal vertebra, intercostal nerves, etc.). In one case pain may have been due to esophageal stricture rather than to aortal pressure. In general, the elongated contour of the aneurismal dilatation would in itself argue against pressure upon, or erosion of, surrounding tissues. The rich nerve and ganglionic plexus surrounding the root of the aorta, and the nerve fibers and isolated nerve cells which have been described in the connective tissue of its middle coat, may explain how various grades of inflammation in the aorta and how differences of aortal pressure and dilatation can give rise to these referred pains. In addition, periaortitic inflammation with possible involvement of the neighboring nerve structures can also cause neuralgic symptoms. Similar nerve involvement has been found in dilatations of the ascending aorta and arch. The assumption of inflammatory exacerbations within or without the aorta is corroborated by the occasional rapid subsidence of the pains following salvarsan injections. This result is probably ascribable to control of these exacerbations, although Vaquez and Laubry, and Vaquez and Bordet claim that there is sometimes a reduction in the size of sacculated aneurisms after several salvarsan injections. The writer has not been able to determine any difference in the size of the dilated descending aorta as the result of therapy. In addition to the aortal disease it must be remembered that accompanying coronary sclerosis and myocarditis can also produce cardiac pains.

Another group of symptoms is that due to **cardiac decompensation**. This is not necessarily a marked clinical feature; in fact, in three of

the cases it consisted only of slight dyspnoea upon exertion. Its presence seems due to accompanying cardiovascular disease rather than to the aortic dilatation itself. Edema is usually slight and confined to the legs; it is extreme only in neglected cases or late in the disease. Dyspnoea is of the usual cardiac type; it is generally most marked on exertion; it is continuous in the severe cases with cardiac failure. Left ventricular hypertrophy of varying degrees is usually present. The heart is occasionally tremendously hypertrophied before cardiac failure sets in.

Diagnosis. — The diagnostic criteria of aneurismal dilatation of the **arch** and **ascending aorta** have frequently been emphasized, particularly the rough systolic murmur and the accentuated, ringing second sound at the right base. In dilatation of the **descending aorta** abnormal sounds in typical instances are best heard at the **left sternal border at its middle third**, or from the third left intercostal space to the ensiform. This is approximately the site of the projected dilatation upon the chest wall. The area of propagation of the murmurs is somewhat similar to that occasionally found in valvular aortic regurgitation. The signs outlined — the impact area, the rough first and accentuated or liquid second sounds — will usually serve as differential guides. There is in addition, in the aneurismal dilatation described, a rough systolic murmur over the dilated aortal area. The second sound has a liquid rather than an accentuated tone, and is prolonged through the entire diastole or is followed by a diastolic murmur of varying intensity. Although this double murmur is the rule, the only auscultatory difference may be a slightly impure first and a somewhat accentuated second sound. By placing the eye upon a level with the patient's chest a distinct heaving area distinguishable from that at the apex, and occupying the lower left sternal intercostal spaces, can often be detected. A sensation of impact is also given to the bell of the stethoscope when, during the usual clinical examination, it is placed over this area. This impact sensation is particularly well detected by snugly pressing two or three fingers in the left middle interspaces near the sternum. Occasionally a systolic thrill is also palpable. All these signs are made more evident by having the patient hold his breath at the end of expiration. In addition an interval in the time of thrust between the apical and left intercostal area is sometimes noted by placing one finger over the apex and another over the left sternal border. Since, however, this interval, physiologically, comprises .07 second, it may be impossible to distinguish it by palpation. Another method for detecting this difference is the use of a differential stethoscope, *i.e.* an ordinary pair of ear tubes arranged with two bells, one over the apex and the other over the dilated aorta. When the murmurs are not too loud, the difference in time impact can thus be determined. The writer has also attempted to establish this difference by placing two receiving cups of a polygraph over these areas and noting the time of arrival of the thrusts by the writing pens upon the polygraphic paper, but the results were inconclusive.

Differential Diagnosis. — It is important to distinguish the aortic impacts to the left of the sternum from those found in patients with marked left ventricular hypertrophy or in healthy individuals with overacting hearts and thin chest walls. However, the auscultatory signs above described are then absent. Although there are many refinements of percussion methods used in the attempt to delicately outline the dilated arch and ascending aorta (for example, threshold and auscultatory percussion, Chapter X) their value in dilatation of the descending aorta seems exceedingly problematical, because the aorta is deep seated and most of the enlarged area is situated behind the ventricles. An examination of the posterior chest wall also fails to reveal any difference from the normal physical signs. In three of my cases in whom a correct tentative diagnosis was made before fluoroscopy, all methods of percussion failed to reveal any enlargement of the descending aorta. To clinch the diagnosis, examinations by means of the fluoroscope or roentgenograms are essential. As in examination for suspected disease of other portions of the aorta, the patient should be fluoroscoped in several lateral positions. Fluoroscopy must be practiced carefully in order to reveal and distinguish the darker silhouette of the dilated descending aorta behind the left ventricle. Roentgen-ray plates must also be carefully scrutinized for the same reason. It is important in this connection to again indicate the differences in shadow areas between orthodiascopic fluoroscopy and roentgenograms of the heart and aorta. It has been my experience that the former produces very little distortion in the size of the cardiac and aortal areas, because the X-ray tube and screen move together and thus approximately parallel rays reach the observer. In the roentgenogram, in which the rays are always divergent, there is an increased cardiac shadow. Comparisons between the orthodiascopic tracings and roentgenograms in my series of cases occasionally showed marked discrepancies in the size of the dilated aorta and heart.

Therapy. — Treatment may be conveniently divided into three parts: that of the underlying disease, of the decompensation, and of the pains. The majority of cases of aortic disease are known to be of luetic origin, the Wassermann reaction being positive in most instances. Salvarsan was originally considered contraindicated by Ehrlich in cardiac lues because of the fear of overwhelming the system with spirochetes (Herxheimer reaction); but experience has shown that the drug judiciously administered is definitely indicated in this disease. Three of the cases here reported were thus treated with excellent results. The best routine procedure is the intravenous injection of .2 gm. of salvarsan every week until three doses have been given; then, if indicated, it may be repeated in .6 gm. doses a month or two apart. In the interim, intramuscular injections of mercury about twice weekly should be given in conjunction with iodide of potash. If the luetic changes in the aorta are such that calcification and scar-tissue formation

are extensive and the myocardium is the seat of advanced disease, the treatment can naturally be of little or no avail. The degree and extent of such pathological changes cannot be diagnosed accurately enough by our present methods, although Vaquez and Bordet claim to have observed with the fluoroscope calcified areas in the ascending and transverse aorta, and the diminution of these areas after salvarsan injections. Since the treatment outlined is often efficacious and is followed by marked improvement, I believe it should be carried out in all cases unless the patient is *in extremis*. Even if the Wassermann reaction is negative, the same therapy should be instituted, because syphilis is the preponderatingly etiological factor of extensive aortitis, and because in any case salvarsan is not followed by serious results. Besides, a Wassermann reaction which upon the first examination is negative may later become positive even though no provocative injections had been given; this happened in one of my cases.

Cardiac failure accompanying dilatation of the descending aorta requires the same treatment as that from any other cause. A reliable preparation of digitalis should be given. I prefer the tincture administered undiluted in 15-drop doses three times daily. If the case is urgent and very little or no digitalis had previously been given, 1 cc. of a 1 per cent solution of strophanthin can be slowly injected intravenously. The objection that digitalis in therapeutic doses raises blood pressure has been sufficiently disproved by recent careful investigations (Chapter XVI). Digitalis had no effect upon the blood pressure in any of my cases; the pressure was as often lowered as raised during its administration. Symptomatic treatment of the pains occasionally requires codein or morphine, but they are frequently relieved by the antiluetic treatment which acts by controlling the inflammatory exacerbations of aortitis and periaortitis.

Prognosis. — Dilatation of the descending thoracic aorta is frequently overlooked; in fact, except in the course of a routine roentgen-ray examination of the chest, its diagnosis by ordinary clinical methods has, to my knowledge, never previously been made. If the patient seeks advice before severe decompensation sets in, and if then the condition is correctly diagnosed and vigorous antiluetic treatment instituted, the patient may live in comparative comfort, for the aortal disease is an index of the general cardiovascular mischief rather than in itself the cause of cardiac failure. Of the three patients with slight symptoms who have been under observation for one year or more, two are clinically well and one much improved.

Cure, in the sense of a return of the aorta to its normal state, is impossible; but, as in other organs the luetic disease can be arrested and controlled, so the heart and aorta, although somewhat crippled, may be sufficiently restored to make the patient comfortable. Rupture of the dilated ascending and transverse aorta is extremely infrequent. Unless the luetic disease is confined to a small area, this accident seems

less likely to occur in the descending aorta because of its greater length and because the dilatation occupies a larger area.

Because of its importance I shall **summarize** the clinical complex of aneurismal dilatation of the descending aorta as I have observed it. The Wassermann test was done in four of the five cases. It was positive in two and negative in two. One of the latter gave a definite history of luetic infection. Three cases had slight symptoms when treatment was begun; these were clinically cured. One, with severe heart failure, was much improved. In three the correct diagnosis was made by the presence of an impact area to the left of the sternum at its middle third, and by prominent localization of the murmurs over this area. Electrocardiograms were taken in four cases; three showed complexes of left ventricular preponderance, the fourth was normal. The physical signs of all the cases were most marked when the symptoms — dyspnoea, pain, or cardiac failure — were present; the signs became less with improvement.

In conclusion it may be stated that aneurismal dilatation may be confined to the descending thoracic aorta alone; it may then occasion sufficiently definite physical signs to lead to its tentative diagnosis by the ordinary methods of clinical examination. The physical signs are most prominent to the left of the sternum at its middle third. Roentgen-ray investigation is indispensable for a positive diagnosis. The symptoms are often very slight. The disease may run a mild course, lasting many years. Intensive antiluetic treatment — salvarsan, mercury, and iodide of potash — is indicated in every case.

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CHAPTER XIV

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CHAPTER XV

SYMPTOMATOLOGY, PHYSICAL SIGNS, DIAGNOSIS AND PROGNOSIS OF MYOCARDITIS AND OF CARDIOSCLEROSIS

FROM a description of the pathological changes found in **myocarditis** (Chapter XII), it becomes apparent that the symptomatology must be a varied one, and, to a great extent, must depend upon the type and extent of the pathological process.

It is important to remember that the diagnosis of myocarditis, when present alone, is often extremely difficult or even impossible. Often, the diagnosis must be made from the symptoms of myocardial insufficiency. Myocarditis rarely exists as an isolated pathological lesion. It is usually combined to a varying degree with the pathological ensemble characteristic of **cardiosclerosis**, the physical signs of which are much more definite and more readily recognizable than those of myocarditis alone.

The physical signs of myocarditis as an isolated entity may be limited to some evidence of enlargement of the heart, or, in advanced cases, of severe degeneration, to weakness of the first sound at the base and apex, and to weakness or entire absence of the second sound, especially at the base. From the physical examination alone it may not be possible to venture more than a guess as to the extent, or even the presence, of any myocardial damage. When considered, however, in conjunction with such other data as furnished by the etiological factor, the size of the heart, the condition of the palpable arteries, the absence of valvular disease, the presence of muffled and indistinct cardiac sounds, moderate tachycardia with dyspnoea, the subjective and objective phenomena indicative of myocardial insufficiency, it may be possible to state, with a fair amount of accuracy, the extent of myocardial mischief. It is clear, then, that the diagnosis must often be established inferentially and by exclusion.

With reference to **cardiosclerosis**, if the morbid process is at all advanced, it is readily diagnosed from the physical signs. Prominent among these are the signs of aortitis. If the latter is accompanied by simple or aneurismal dilatation of the arch of the aorta, one often finds, on **inspection**, a broad expansile rise of the tissues in the supra-sternal notch, due to the vigorous overaction and enlargement of the

arch. In advanced cases of arteriosclerosis the carotid arteries may be tortuous and thickened and may pulsate very vigorously and with an accentuated thrust. If the first part of the aorta is especially involved in the degenerative process, the carotid phenomenon may be more marked on the right than on the left side. In a thin-chested individual, with the patient lying prone, and with the eye of the observer on a level with the patient's chest, it is sometimes possible to see the tissues over the right second interspace pulsate in simple or aneurismal dilatation of the aorta. The fluoroscopic and orthodiascopic aspects of these lesions (Chapter IX), and the difficulty of mapping out the aortal limits by physical examination (Chapter X), have already been described.

Corresponding to the physical signs discovered by inspection, on **palpation** there is a marked thrust felt by lightly insinuating the finger behind the sternum; a similar thrust may be found over the carotids. Systolic expansion over the right base may likewise be felt by snugly applying the fingers laid flatly in the second and third right interspaces near the sternum.

The **auscultatory signs** of typical aortitis are characteristic and readily recognized. The first sound is rough and harsh; the second, accentuated, and, in some instances, metallic and tinny in character. The latter has usually been interpreted as due to hypertension, a frequent accompaniment of aortitis. It is fairly frequent, however, when hypertension is not present. Acoustically, it seems probable that the metallic tone is at least partly due to dilatation of the aorta, as already described (Chapter XIV). The roughened and harsh first sound is occasionally heard by placing the stethoscope in the jugulum over the pulsating arch; this phenomenon depends partly upon the extent of aortic enlargement and partly upon the height of the aortic arch. The murmur is sometimes propagated along the carotids. Following the second sound, a diastolic murmur of varying duration and intensity may be heard over the right base. It is usually transmitted downwards along the right sternal border; it is on rare occasions heard best over the third left interspace and along the left sternal border.

Although the physical signs above described are characteristic and typical of aortitis, there is scarcely a cardiac lesion which produces more varied or atypical auscultatory phenomena. For example, the sounds may be perfectly normal at the right base, the first or second sounds or both may be extremely faint or entirely absent. Upon what changes in the aorta such differences depend it is at this time impossible to state.

The sounds that are heard at the apex are rarely significant in the diagnosis of aortitis. They apparently vary with the state of efficiency of the circulation, and, to a lesser extent, with the state of hypertension. For example, the first sound may be unclear and muffled, or entirely absent. Occasionally, in cases of hypertension, with or without left ventricular hypertrophy, the second sound at the apex takes on char-

acteristics similar to that at the right base, *i.e.* it becomes metallic in tone. In conjunction with a weak or absent first sound, the cardiac impact may be weak or scarcely palpable. One instance characteristic of these findings I was able to prove at necropsy. The patient complained of severe epigastric pains, especially on walking. The cardiac examination showed a muffled first sound over the right base and a scarcely perceptible first sound at the apex. Polygraphic tracings showed heart block. The orthodiascopic tracing showed enlargement of the first part and arch of the aorta, and an enlarged left ventricle. The patient died suddenly. At necropsy, there was diffuse dilatation and thickening of the aorta from its root to, and including, the upper abdominal aorta; spirochetes were isolated from the aortal wall. There was moderate left ventricular hypertrophy; the myocardium was riddled with scar tissue.

While physical signs like those I have described are suggestive of myocarditis and aortitis, these lesions can be present and yet sounds at the apex and base be perfectly normal. A faint systolic murmur may accompany the first sound; this, however, possesses no diagnostic value, for such impurities are by no means rare in normal hearts. Of much more significance is the presence of a loud and rough systolic murmur heard most prominently at the apex, and sometimes transmitted to the left. These murmurs are probably caused by fibrous or calcareous valvular thickening (Chapter XII) with consequent mitral regurgitation (Chapter X). In old individuals with marked senile arteriosclerosis and widespread aortic and valvular changes—a cardiovascular condition also found occasionally in the adult—there is sometimes heard a very loud, rough, systolic murmur over the entire anterior part of the chest; this murmur is transmitted to the left, and may even be heard posteriorly between the spines of the scapulæ. In its harshness, loudness, and area of transmission, it is extremely suggestive of aneurism; however, a diastolic murmur is rarely encountered and the roentgenograms of the cases that I have studied have not regularly shown abnormal aortic enlargement. This loud systolic murmur is apparently the result of the coalescence of two components: an aortic systolic and a mitral regurgitant murmur. The aortic factor is either atheromatous change in the aortic flaps producing stenosis, or the impact of the blood stream over the roughened sclerotic aortal wall; both causes sometimes operate together to produce this aortic component. The mitral component of the murmur is due to sclerotic changes in the mitral cusps. The intensity of the combined murmur seems to depend upon the state of myocardial efficiency. I have found it exceptionally loud only in those who were compensated, or in whom there was but slight cardiac failure. On the other hand, this murmur may be absent in those who clinically or at necropsy show advanced atherosclerotic changes. Such an instance is the following:

A robust man of 44 complained chiefly of substernal pains upon

slight exertion. There were occasional attacks of nocturnal dyspnoea. The only abnormality upon auscultation was slight muffling of all the cardiac sounds. Several Wassermann blood tests were negative. The patient finally died of myocardial insufficiency. At necropsy, incrustated calcareous deposits were found on all the valves. The coronaries were thickened and impermeable, the aortal arch was markedly atheromatous, the myocardium contained many fibrous patches. As already suggested, the absence of loud, harsh murmurs in this case was probably due to failing circulation.

An extremely valuable hint regarding the degree of cardiosclerotic change is sometimes given by the presence of pericardial adhesions. These are found most frequently over the right base or in the apical region. Superficial creaking sounds or pericardial rubs are then heard at the end of long inspiration, or accompanying cardiac systole. The importance of diagnosing these old localized adhesions lies not so much in the fact that they add embarrassment to the circulation, but because they are found only when the cardiosclerotic changes are very advanced.

Thus far, most stress in the diagnosis of cardiosclerosis has been laid upon the auscultatory signs. Other important diagnostic data may be gained by examination for ventricular hypertrophy, hypertension, and palpable and audible reduplicated first sounds at the apex. The significance and bearing of all these phenomena are more fully described in their appropriate connections.

Symptomatology of Cardiosclerosis.—As the morphology of cardiosclerosis is a varied one, so its symptomatology depends upon the factor that is clinically predominant. It is evident that it is often impossible to group these patients into one class, for the symptoms produced by the various underlying pathological changes overlap. It has already been pointed out, for example, that arteriolar and capillary fibrosis may be a generalized pathological process, or may attack individually the arterial system of the heart, the kidneys, or the brain. In such types, the symptoms are respectively predominantly cardiac, renal, or cerebral. We shall here concern ourselves only with those individuals in whom the cardiac symptoms form the chief complaint. As with other cardiac patients, the earliest subjective symptom is usually dyspnoea. Its onset is gradual and evident at first only after severe exertion. Later, the dyspnoea occurs upon slight exertion. It is frequently associated with a sense of weight or oppression on the chest, at first transient, and later constant. These sensations must be differentiated from the precordial pain and distress (Chapter XXI) from which such patients also suffer. Precordial pains may be slight and localized, or severe and radiate to the left axilla and arm, to the neck, interscapular region, or occasionally to the right arm. At first the pains occur after exertion; later, they may appear in attacks which rouse the patient from a sound sleep. Slight hemoptyses occurring after exertion sometimes antedate the subjective symptoms for months or

even years. An enlarged, congested liver, smooth and globular in outline, is another objective symptom which may be found before the patient complains of dyspnea. It is not uncommon to feel the edge of the liver several inches below the free border of the ribs. Epigastric sensitiveness to pressure is fairly frequent. It is found even when the liver is not enlarged. The cause is usually assumed to be congestion of the gastric mucous membrane; in another connection, I have pointed out that it is probably of reflex origin, the source primarily being excitation of the cardiac nerves.

Pulmonary examination often reveals crepitant râles at both bases. Their presence is prognostically suggestive of subsequent attacks of pulmonary edema. The latter may be mild and recur frequently, or a very severe attack may suddenly take place after unusual exertion or excitement. Hydrothorax, usually right-sided, may also be present. Edema of the legs is, at the outset, only slight, and is usually present after the patient has been up and about, unless the process is far advanced and has not responded to therapy.

The types of **arrhythmias** encountered in cardiosclerosis are of some clinical significance. Those found in senile cases will be discussed later. When decompensation is not far advanced, the pulse is regular. Extrasystoles usually represent the first type of arrhythmia to appear; they are mostly of the ventricular variety. The next most frequent type is auricular fibrillation. Heart block is usually found only in advanced cases. In isolated instances, attacks of paroxysmal tachycardia alone, or alternating with auricular fibrillation, are found. In one case, for example, a man of 54 with moderate hypertension, mild cardiosclerosis, and slight precordial pain, there was an attack of paroxysmal tachycardia lasting one week; thereafter the patient felt well for several months. He then developed auricular fibrillation, again accompanied by only slight precordial discomfort. This attack lasted four days. The patient was feeling quite comfortable, and his condition was apparently progressing favorably. While sitting in a chair the patient suddenly died. In neither attack was dyspnea severe or a prominent symptom. In some patients the cardiac irregularities change suddenly, and apparently capriciously, from one type to another, especially from extrasystoles to auricular fibrillation, or vice versa. An example has just been quoted. Such variability I have found of ominous prognostic import.

There exists a small group of patients in whom digitalis does not relieve decompensation, but produces diverse arrhythmias in rapid succession; these are chiefly sinus arrhythmia and extrasystoles, either isolated or coupled. Here, too, I have found that the occurrence of such varied arrhythmias may portend a fatal outcome within a few weeks or months.

In patients with **senile cardiosclerosis**, — that is, those who have in addition to cardiosclerotic changes palpably thickened and tortuous

arteries, — **auricular fibrillation** is very common. One of my hospital services consists of old people with and without cardiovascular symptoms; among these there is a large proportion of fibrillators. In those who are compensated, the cardiac rate is usually between 70 and 80 per minute, most of the beats being effective and reaching the wrist. In those who suffer from severe decompensation the cardiac activity is rapid and quite irregular, with many frustrane beats. The course of the elderly cardiosclerotic patient is essentially afebrile, unless disturbed by complications. Of these the most frequent consist in attacks of pneumonia, the majority of which are of hypostatic or embolic origin.

PROGNOSIS IN CARDIOSCLEROSIS

Under this caption are included those non-rheumatic chronic cases of heart disease which show various degrees of involvement of the myocardium, and atheromatous changes of the endocardium, the coronaries, the valves, and the aorta. With the exception of syphilis, the bacterial infection or chemical poison has long since reached the quiescent stage; the pathological process, however, in most instances insidiously and slowly continues. Clinical and pathological proof of this is usually lacking, since the slow progress of the damage can scarcely show itself except by careful clinical observation of the same patient over a long period of time, or by occasional necropsies of patients in whom the disease had been watched and studied since its incipency.

The kind and frequency of **complications** found in cardiosclerosis are, in the main, different from those in rheumatic endocarditis. Prognostically, much depends upon the type of disease which dominates the clinical ensemble. For example, if the uremic element predominates, the prognosis will revolve upon that; cerebral hemorrhage or acute uremia are then apt to be the commoner ways in which the disease will terminate. If arteriosclerotic, the terminal accidents are prone to be hemiplegias or monoplegias. But the arteriosclerotic syndrome itself can occasionally be clinically subdivided into that affecting the aorta and the coronary circulation, and that involving the myocardium; the prognosis must then be studied from these separate standpoints.

General Prognosis. — 'Acute accidents' which may occur and which naturally immediately change the entire prognostic picture will be discussed later. Aside from these, the general prognosis depends upon an attempt to gauge the rate of development and extent of cardiosclerotic damage, and the actual degree of myocardial insufficiency. The first is only derived from careful and patient inquiry into the onset of cardiovascular symptoms and from tangible evidence of an underlying infective process. From these the duration of the disease may be estimated. For example, in one instance of severe myocarditis with decompensation in a physician of 55, I was definitely able to

establish the commencement of the disease as a mild nephritis following grippe infection some 20 years previously. This the patient had entirely overlooked as being of no etiological significance. Very slight cardiac symptoms began 15 years ago. They consisted of slight dyspnoea and occasional hemoptyses. Acute symptoms of decompensation appeared only a few months ago; they usually occurred after rapid walking or after coitus. The fact that cardiac symptoms began 15 years ago, and the present status of widespread cardiosclerosis as revealed upon examination, make it appear that the pathological damage must have long antedated the symptoms. The entire history speaks for a steady though very gradual continuation of the pathological mischief. This case well illustrates the meaning conveyed, and the information to be gained, by a careful clinical history, in an attempt to roughly approximate not only the degree of cardiovascular disease but the length of time required to reach the condition found when the patient first presents himself for examination. The degree of myocardial insufficiency is gauged not only by physical signs, but also by its usual clinical manifestations; these are chiefly dyspnoea, precordial distress, and a sense of exhaustion after effort.

It will be noted that the amount of cardiac hypertrophy and of hypertension have not been emphasized because they are more concerned with sudden heart failure and cerebral accidents than with the question of 'general prognosis.' Thus, specimens of extremely hypertrophied and diseased hearts have been removed from patients who lived to ripe old age, and in whom the disease had doubtless been present for many years. So, too, clinicians have for many years followed patients who had severe aortal and myocardial involvement, some of them proved as such at necropsy; yet during this long period there may have been no marked discomfort and almost negligible subjective symptoms from the cardiac disease.

With our present knowledge, we cannot state why some tremendously hypertrophied hearts carry their circulatory burdens fairly well for many years, while other apparently more nearly normal hearts suffer from circulatory failure. I would suggest that the activity and degree of the progress of the pathological process play important rôles. The system may accustom itself to a gradual curtailment of its circulatory reserve, while a more acute though less widespread pathological change might be attended by quick cardiac exhaustion in a heart already working at or near its maximal energy.

In order to study **myocardial insufficiency** from the standpoint of prognosis, it is necessary to estimate not only the amount of gross pathological damage to the cardiovascular system, but also the amount of fairly healthy tissue sufficient to carry on the circulation. For these purposes, besides careful physical examination, it is necessary to examine the cardiac reserve power so as to determine whether any "factor of safety" remains. Functional efficiency tests (Chapter XVIII) may be of

some value in this connection. Of more aid and more readily applicable are the facts already mentioned, which can be determined by the ordinary methods of clinical examination. These include the investigation of the result of effort and exercise upon the heart rate, the patient's subjective sensations, the size of the liver, the presence of dyspnoea, edema, hydrothorax, and precordial distress.

To study ideally the degree and rate of progress of the pathological damage requires a knowledge of the patient over a number of years, but this opportunity is only exceptionally vouchsafed us. We shall therefore have to depend in the main upon the data and history furnished by the patient. Of great importance prognostically are the frequency, type, and duration of attacks of decompensation, and the manner and rapidity with which they yield to proper therapy. These considerations are often glossed over or entirely disregarded; yet from them, in addition to other data, one is frequently correctly guided as to the ultimate 'general prognosis.' Much will naturally depend upon the individual opinion of what is "proper therapy." If, for example, decompensation has been relieved after a few days' rest and dosage with a reliable preparation of digitalis, or if edema has been relieved and diuresis promoted by diet (*q.v.*) and some caffeine derivative (theobromin, theocin, caffeine), it is safe to make a good general prognosis regarding longevity. On the other hand, if decompensation is frequent, marked, and refractory to treatment, the general prognosis becomes correspondingly poor.

Knowledge of the prognostic features included under the term **Acute Accidents** depends upon the fact that, in the pathology of cardiosclerosis, there is an admixture of myocardial, arterial, and valvular disease in varying proportions; it requires not only laboratory tests and detailed examination, but also clinical acumen and broad experience to decipher and properly apportion the damage to the heart, kidneys, arteries, and brain. Thus, aneurismal dilatations of the aorta may be confined to its first portion, the arch, or to the descending thoracic aorta, and not markedly affect the coronaries or myocardium. In other patients with cardiosclerosis, the coronaries and their branches are only slightly involved, while the brunt of the disease affects the myocardium and produces myofibrosis and hypertrophy. Conversely, coronary disease may be widespread and include subsidiary branches of the second and even third order, yet the myocardium remain comparatively healthy. Again, an arteriosclerotic process may spend itself chiefly upon the circle of Willis, and produce symptoms of cerebral softening. All these instances show how necessary it is to search out the lesion which is symptomatically and pathologically predominant; for in this manner alone can a fairly scientific prognosis be grounded, and 'acute accidents' foretold and possibly guarded against.

For the purpose of studying acute accidents **prognostically** patients with cardiosclerosis may be roughly grouped into those with predominant arteriosclerotic, uremic, or cardiac symptoms. In cases of general

atheroma without marked hypertension (usually found in the senile), the commoner sudden causes of death are cerebral hemorrhage, pneumonia, or pulmonary stasis. The latter produces bubbling or crepitant râles at both bases. These signs may be present for weeks or months before the acute accidents occur; they help to presage probable pulmonary edema or a terminal pulmonary infection.

A fairly definite hint of cardiac failure or of impending death is furnished by the presence of arrhythmias, the type of which change, apparently capriciously, hourly or daily. Thus, at various times, extrasystoles, auricular fibrillation, or tachycardia may alternate with a regular pulse. This heightened ventricular irritability is not necessarily associated with any demonstrable change in the clinical symptoms. It seems to be due to some profound change in cardiac nutrition dependent upon severe sudden focal myocardial damage from emboli or infarcts. The following cases will serve to illustrate the clinical picture.

Male, age fifty-five, was suffering from mild nephritis and cardio-sclerosis with moderate hypertension. The main complaint was slight dyspnoea upon climbing stairs. He improved under therapy and felt comparatively well for one year. His pulse had always been regular until one day he complained of constant "fluttering" in his heart. Clinical examination and electrocardiograms showed a simple tachycardia; the rate was 160 per minute. His condition was accompanied by very slight dyspnoea. There was no precordial pain. The tachycardia lasted two days. For a period of three months he again felt well, his pulse remained regular. Then, suddenly, during the night he complained of feeling faint and of very slight "uneasiness" in the chest. Again slight dyspnoea was present. Examination showed auricular fibrillation with rapid and irregular heart action; the cardiac rate was 150 per minute. He was put upon digitalis and later was allowed to sit out of bed. The fibrillation continued, but the patient said he felt very much better. Despite apparent improvement and absence of pain, a diagnosis of probable coronary infarct was made, and a guarded prognosis given regarding the outcome of the attack. After four days, while the patient was sitting quietly at the table and reading, he gasped and died within two minutes.

Female, age sixty-seven, well preserved, had been complaining of substernal pain for one year. More recently, she began to have attacks of dyspnoea and of pains lasting one hour and radiating to the back. At such times, the pulse became "irregular" (auricular fibrillation?); between attacks it remained regular. Upon examination, the systolic blood pressure was 168, the diastolic 80; there was slight pretibial edema. Fluoroscopic examination showed a somewhat dilated aortic arch. The urine contained neither casts nor albumen. The electrocardiographic tracing showed a few ventricular extrasystoles. A diagnosis of probable coronary sclerosis was made and sudden death indicated, mainly because of the arrhythmia which accompanied

the attacks of dyspnoea. Two months after the first examination, she had an attack of dyspnoea and precordial pain, and died within a few minutes.

Prognosis in Cardiosclerosis with Coronary Disease.—Where disease of the coronaries is assumed to be the main cause of the symptoms (Chapter XXI), the prognosis depends upon the severity of the precordial attacks and upon the extent of concomitant cardiac damage; in other words, upon the state of preservation of the remainder of the cardiovascular apparatus. If compensation is good and the precordial distress is mild and yields to therapy, the patient may continue a fairly comfortable existence for years; in such cases, fair but somewhat guarded prognoses regarding longevity may be given. Widespread coronary disease may show itself clinically in several ways: by severe attacks of precordial distress (angina pectoris), in any of which the patient may die from edema of the lungs, coronary infarct, or embolus; by attacks of dyspnoea; by vomiting which has no relation to the ingestion of food and is often ascribed to indigestion and hyperacidity; or by slight febrile disturbances combined with tender areas over the precordium. I believe the latter is sometimes due to focal myocardial or even to myomalaceous areas from an infective or embolic process in a secondary coronary vessel. Such a process may run its course in a few days or weeks; the heart becomes permanently affected, but the damage is not necessarily incompatible with life or with a fair amount of comfort. The prognosis depends upon the severity of the infection as judged by the temperature, rigors, duration, and, especially, by the frequency of the attacks. When the clinical manifestations are mild, with only occasional recurrences, the prognosis is fair, for the chances are that the myocardial damage occurring with each infection will only gradually cripple the heart, and that symptoms of myocardial insufficiency will supervene only after a long period of time. The chief danger is the possibility of embolic infarcts into some vital organ; for example, the brain.

The **main right or left coronary** artery may be suddenly plugged by an embolus, or its lumen closed by an infarct. This is usually accompanied by intense precordial distress, dyspnoea, and gastric symptoms. Signs of acute cardiac failure rapidly supervene, and death usually occurs within 24 hours. Such cases have been corroborated by necropsy examinations.

Ventricular fibrillation is probably another type of sudden death occurring in patients with cardiosclerosis. As far as our knowledge goes, this arrhythmia is apparently almost immediately fatal.¹ At present there is no method by which its advent can be foretold. It seems most likely to occur in patients who receive digitalis (Chapter XVI) and who, just preceding the presumed fibrillation attack, are in a fair state of compensation and apparently progressing favorably.

The **degree of cardiac hypertrophy** has a certain influence in increasing the probability of 'acute accidents,' especially after a first attack

¹ Robinson, however, recently reported a case in which the patient lived several weeks after the onset of ventricular fibrillation.

of decompensation. The cardiac reserve power is then apt to be quickly exhausted, even after the restoration of compensation. This may evince itself by the presence of dyspnoea, slight edema, visceral congestion, or by arrhythmias, especially extrasystoles. The usual agonal stages consist in edema of the lungs coming on in attacks, or in steadily increasing pulmonary engorgement.

Prognostically **hypertension** forms an extremely important factor in the possible causation of cerebral hemorrhage. There is, however, no definite method of foretelling these apoplectic attacks. In many cases the blood pressure is high for months or years, and cerebral hemorrhage does not supervene; in others, hypertension is only moderate and hemorrhage does occur. The reason for this probably lies in the fact that this accident depends not only upon the degree of hypertension but also upon the extent of the cerebral arteriosclerosis. In the absence of brain symptoms, we possess no means of determining the extent of cerebral arteriolar disease.

Uremia is another frequent form of death in cardiosclerosis. It has been found that the diastolic pressure is of great significance in determining the probability of uremic complications. A diastolic pressure of 110 mm. or over is regarded as a danger signal; the higher the diastolic, the greater the chances for uremia, with all its symptoms and complications. Briefly, these are vomiting, nausea, headache, oliguria, dyspnoea, and pallor. The special prognostic features of each of these manifestations are beyond the scope of our subject.

PHYSICAL SIGNS OF VENTRICULAR HYPERTROPHY

The possibility of correctly diagnosing cardiac hypertrophy by the usual physical signs depends chiefly upon the extent of the hypertrophy and the thickness of the chest wall. For example, the tremendous impact to the thoracic wall in the far advanced and extreme ventricular hypertrophy of typical aortic insufficiency can scarcely be mistaken, for it produces a visible and palpable shock to the entire left half of the chest. It is the lesser degrees of cardiac hypertrophy which especially require diagnostic consideration. Hypertrophic and enlarged hearts, in which myocarditis is an important pathological factor, are often not diagnosed because the impaired condition of the circulation is such that the usual vigorous, unhampered action indicative of hypertrophy is absent. The diagnosis must then depend upon the data already indicated in the description of the physical signs of myocarditis (*q.v.*).

When typical, **left ventricular hypertrophy** produces a booming, loud, first sound over the entire cardiac area, and a broad, heaving systolic impact distinctly palpable well outside the normal cardiac limits. It must be remembered, however, that these characteristics may be simulated in thin-chested individuals in whom the heart is well applied

to the chest wall, or by hearts in which the systolic action is violent (for example, in exophthalmic goiter). Such instances demonstrate the necessity for reserve in the diagnosis of ventricular hypertrophy, particularly in view of the fact, already pointed out, that it is extremely difficult to delimit, even approximately by percussion, the size of the cardiac area. In other words, the physical evidence of cardiac enlargement must be definite and unmistakable before its diagnosis is ventured. An accentuated second sound at the apex or at the second right interspace is sometimes accepted as an indication of left ventricular hypertrophy. It is true that these abnormally accentuated sounds are then often present, but they must be regarded not as evidence of hypertrophy, but rather of hypertension in which left ventricular hypertrophy is often, but not invariably, present. A reduplicated apical impulse (gallop rhythm) is sometimes present in the hypertensive cases, especially when cardiac hypertrophy exists.

The diagnosis of **right ventricular hypertrophy** is even more difficult and problematical than that of the left. A second pulmonic sound, relatively or actually accentuated when compared with the second aortic, is regarded as evidence of overstrain of the right heart, and hence indicative of probable right-sided hypertrophy. However, I have observed moderate accentuation of the pulmonic second sound very frequently in children and adults with normal hearts in whom tachycardia existed. I believe that a comparative study of this sound with the aortic is of value as an indication of probable impairment of the right ventricle only if the accentuation is pronounced and tachycardia is absent. Even then the question of hypertrophy must rest upon the length of time during which the accentuation has been observed, the type of lesion, and upon other associated data. In marked right ventricular hypertrophy the apex may be pushed to the left, so that the anterior surface of the heart consists chiefly or entirely of the right ventricle. Furthermore, the cardiac area of dullness may not be increased to the right because the right ventricle normally lies mainly upon the diaphragm. Marked visible and palpable epigastric pulsation and systolic retraction of the apical impulse are the physical signs upon which most stress is laid in the diagnosis of right ventricular hypertrophy. In a series of cases in which the ventricles were separately weighed at necropsy, Lewis did not find either visible or palpable epigastric pulsation or thrust in a sufficient percentage of cases to make these signs of diagnostic value. These are examples of the difficulties with which the diagnosis of right ventricular hypertrophy is surrounded.

It is thus evident that, except for massive ventricular hypertrophy, the clinical diagnosis of left or right ventricular hypertrophy from physical signs alone is often problematical, and that in many instances its presence can only be correctly inferred in conjunction with the known pathological lesion, — valvular, myocardial, or nephritic.

of decompensation. The cardiac reserve power is then apt to be quickly exhausted, even after the restoration of compensation. This may evince itself by the presence of dyspnoea, slight edema, visceral congestion, or by arrhythmias, especially extrasystoles. The usual agonal stages consist in edema of the lungs coming on in attacks, or in steadily increasing pulmonary engorgement.

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PHYSICAL SIGNS OF VENTRICULAR HYPERTROPHY

The possibility of correctly diagnosing cardiac hypertrophy by the usual physical signs depends chiefly upon the extent of the hypertrophy and the thickness of the chest wall. For example, the tremendous impact to the thoracic wall in the far advanced and extreme ventricular hypertrophy of typical aortic insufficiency can scarcely be mistaken, for it produces a visible and palpable shock to the entire left half of the chest. It is the lesser degrees of cardiac hypertrophy which especially require diagnostic consideration. Hypertrophic and enlarged hearts, in which myocarditis is an important pathological factor, are often not diagnosed because the impaired condition of the circulation is such that the usual vigorous, unhampered action indicative of hypertrophy is absent. The diagnosis must then depend upon the data already indicated in the description of the physical signs of myocarditis (*q.v.*).

When typical, **left ventricular hypertrophy** produces a booming, loud, first sound over the entire cardiac area, and a broad, heaving systolic impact distinctly palpable well outside the normal cardiac limits. It must be remembered, however, that these characteristics may be simulated in thin-chested individuals in whom the heart is well applied

to the chest wall, or by hearts in which the systolic action is violent (for example, in exophthalmic goiter). Such instances demonstrate the necessity for reserve in the diagnosis of ventricular hypertrophy, particularly in view of the fact, already pointed out, that it is extremely difficult to delimit, even approximately by percussion, the size of the cardiac area. In other words, the physical evidence of cardiac enlargement must be definite and unmistakable before its diagnosis is ventured. An accentuated second sound at the apex or at the second right interspace is sometimes accepted as an indication of left ventricular hypertrophy. It is true that these abnormally accentuated sounds are then often present, but they must be regarded not as evidence of hypertrophy, but rather of hypertension in which left ventricular hypertrophy is often, but not invariably, present. A reduplicated apical impulse (gallop rhythm) is sometimes present in the hypertensive cases, especially when cardiac hypertrophy exists.

The diagnosis of **right ventricular hypertrophy** is even more difficult and problematical than that of the left. A second pulmonic sound, relatively or actually accentuated when compared with the second aortic, is regarded as evidence of overstrain of the right heart, and hence indicative of probable right-sided hypertrophy. However, I have observed moderate accentuation of the pulmonic second sound very frequently in children and adults with normal hearts in whom tachycardia existed. I believe that a comparative study of this sound with the aortic is of value as an indication of probable impairment of the right ventricle only if the accentuation is pronounced and tachycardia is absent. Even then the question of hypertrophy must rest upon the length of time during which the accentuation has been observed, the type of lesion, and upon other associated data. In marked right ventricular hypertrophy the apex may be pushed to the left, so that the anterior surface of the heart consists chiefly or entirely of the right ventricle. Furthermore, the cardiac area of dullness may not be increased to the right because the right ventricle normally lies mainly upon the diaphragm. Marked visible and palpable epigastric pulsation and systolic retraction of the apical impulse are the physical signs upon which most stress is laid in the diagnosis of right ventricular hypertrophy. In a series of cases in which the ventricles were separately weighed at necropsy, Lewis did not find either visible or palpable epigastric pulsation or thrust in a sufficient percentage of cases to make these signs of diagnostic value. These are examples of the difficulties with which the diagnosis of right ventricular hypertrophy is surrounded.

It is thus evident that, except for massive ventricular hypertrophy, the clinical diagnosis of left or right ventricular hypertrophy from physical signs alone is often problematical, and that in many instances its presence can only be correctly inferred in conjunction with the known pathological lesion, — valvular, myocardial, or nephritic.

If cardiac failure exists, **complete** heart block in itself is not, I believe, a contraindication to the use of digitalis, for the drug cannot increase the dissociation already present. As an instance: I reported a case of complete heart block in which the cause of the dissociation could not be ascertained during life. Digitalis was administered several times until vomiting resulted. There was no change in the dissociation, the only effect of the drug being a subjective thumping sensation in the chest. In **incomplete** heart block digitalis has been regarded as contraindicated because of the danger of the development of complete block. This conversion has been reported in two cases. One came to autopsy. Lesions involving the conduction system and part of the sino-auricular node were found; but similar pathological changes in an instance of auricular fibrillation and heart block, lasting many years, have been described, and digitalis played no rôle in the arrhythmia. The second case was one of severe long-continued decompensation. Digitalis was given for several days; heart block and auricular fibrillation occurred and continued until death a few days later. These two reports do not offer sufficient evidence that digitalis alone was the cause of the block. In neither case was there clinical evidence of any ill effects from the presumed induction of complete heart block by the drug. It appears to me that the possible danger of changing an incomplete to complete block in digitalis medication may be averted by the judicious administration of atropine. The latter should be given in full physiological doses, administered as frequently as the digitalis but about one half an hour before it; for example, atropine may be given before and the digitalis after meals.

In one of my patients with complete heart block in whom, through a nurse's inadvertence, digitalis had been continued for a long time, auricular fibrillation and coupled rhythm were produced in addition to the dissociation. These arrhythmias were not followed by any demonstrable change in the clinical condition.

Pulse alternation is also occasionally caused by digitalis. Because of its usual serious prognostic import when not produced by drugs, digitalis alternation might *à priori* be considered as a contraindication to further medication. A case has been reported in which alternation and extrasystoles were produced by digitalis; despite this, the drug was continued and the patient showed gradual improvement. The underlying cause of alternation in cardiac failure is not known. When produced by digitalis, it apparently is not dangerous, and in itself does not warrant discontinuance of the drug.

As already indicated, the contradictory effects of digitalis are explicable upon the basis of its two distinct actions: that upon the neurogenic control of the heart, thus affecting the rhythm; and that upon the contractile power. Although these effects are usually coincident, the former may precede the latter by one or several days. In other words, arrhythmias occasionally occur before decompensation is

relieved. I do not regard such early appearance of digitalis arrhythmias as any contraindication to further medication. The circulation is only rarely affected by these irregularities; the drug should be pushed in the usual fashion until the beneficial effects upon the circulation are noted. If then the drug is stopped, the arrhythmias usually soon disappear.

Digitalis Vomiting — Administration of Atropine with Digitalis.

— Digitalis vomiting has been commonly ascribed to irritation of the gastric mucosa, but this scarcely explains the marked variations in susceptibility to the emetic action of the drug. Some patients vomit almost immediately, others only after massive doses have been given. A very suggestive explanation of this phenomena is found in experiments on cats in whom the alimentary tract was removed; none the less, all the accompanying manifestations of emesis occurred when digitalis bodies were injected intravenously. These experiments strengthen the belief that digitalis vomiting is probably due to central nerve action and not to peripheral irritation. This fact is of practical clinical importance because, in susceptible patients, small initial doses of digitalis may be advantageously combined with atropine sulphate in doses of $\frac{1}{150}$ of a grain; subsequently the digitalis dosage may be gradually increased to the usual maximum. In this manner I have succeeded in administering digitalis to some patients in whom even small doses ordinarily produced vomiting. Details of the combined administration of digitalis and atropine are given in the following illustrative cases:

Male, age 60, suffered from chronic bronchitis, dyspnoea, and asthmatic attacks for many years. The patient was cyanotic, physical signs of chronic bronchitis, emphysema, and myocarditis were present, the pulse was completely irregular (auricular fibrillation). The tincture of digitalis and Karrell diet (*q.v.*) were given. At the end of one week he was very much improved; digitalis was discontinued because of vomiting. After two weeks the drug was again given with the same result. On the third administration it was combined with atropine sulphate, grain $\frac{1}{150}$, later grain $\frac{1}{150}$, given subcutaneously. In this manner digitalis was administered continuously for several weeks without inducing vomiting and with excellent clinical results.

Female, age twenty-seven years, had auricular fibrillation, a double mitral lesion, and a decompensated heart. Fifteen minims of tincture of digitalis, three times a day, were prescribed. Upon three occasions, after three days' administration, medication was discontinued because of nausea and headache. On two subsequent occasions digitalis was combined with atropine sulphate; one fiftieth of a grain was given subcutaneously when nausea was already present and continued in doses of $\frac{1}{150}$ grain three times a day. Upon the second occasion, atropine and digitalis were given uninterruptedly for two weeks without nausea. Decompensation was relieved.

Female, age twenty-six, had a rheumatic mitral regurgitant lesion. There were broken compensation and cardiac pains for several months. Tincture of digitalis, one drachm daily in divided doses, was given. Usually after a few days medication was discontinued because of vomiting and headache, and the decompensation which had been temporarily relieved, soon recurred. Caffeine, digipuratum, and tincture of strophanthus were substituted for the tincture of digitalis without effect. Finally the latter was combined with atropine sulphate, grain $\frac{1}{150}$, given internally, three times a day. This was continued in two weekly periods, with interruptions, for many months, and the patient showed improvement. Her appetite remained good. She finally decompensated again and died.

The **preparation** and **dosage** of digitalis have long been matters of dispute. My preference has been for the tincture, or for an infusion freshly prepared from potent leaves. A good tincture kept in a properly stoppered bottle will retain its strength for several months. The objection to the infusion is that it must be freshly prepared and requires large dosage, and is more unpalatable than the tincture. The tincture and infusion are therapeutically alike. I have also found the digipuratum tablet an excellent and reliable preparation; it is standardized in frog heart units. One tablet of one and one half grains is equivalent to fifteen minims of the tincture. It sometimes seems more efficacious than the tincture, especially when administered in chronic cardiac disease.

Variability in absorption is no doubt a large factor in variability of the action of digitalis when given by mouth. Digitalis dosage should be regulated by the degree of cardiac failure and the type of cardiac disease. In general, very little if any good is accomplished by starting with doses very much below the average. The **usual total amount** of the tincture required for full therapeutic effect varies from one to one and one half ounces taken over a period of about one week. The average daily amount is one drachm given in fifteen or twenty minim doses. It is best administered undiluted because it has been found that the admixture of water may interfere with absorption. If urgency demands it, very much larger initial amounts can be safely given. One observer, for example, has given initial doses of several drachms of the tincture with great benefit and no untoward symptoms except occasional nausea and vomiting. In urgent cases I have also often administered the tincture in drachm doses four or five times daily; no ill effects were observed. The usual dose of the infusion is from one half to one ounce given three or four times daily. This amount can likewise be considerably increased if quick effects are desired.

The therapeutic effects of digitalis in patients with **rhythmical heart action** become evident by improvement in the signs of decompensation, especially dyspnoea, edema, and the urine output. In **auricular fibrillation** with a completely irregular and rapid pulse, the

therapeutic action is shown, in addition, by the elimination of the irregular and weaker ventricular contractions, the frustate ventricular activity which produces no pulse beats (so-called pulse deficit). In consequence, ventricular rhythm becomes steadier and slower, and the pulse tends to become correspondingly regular. Some observers advise discontinuance of the drug or decrease of its dosage when this result has been achieved. This advice should be followed only if the drug had produced nausea and vomiting, if decompensation had been relieved, or if the usual amount of digitalis (about one ounce of the tincture) had already been taken; for it occasionally happens that the typical discordant arrhythmia of auricular fibrillation comes under sudden control after a few doses, while signs of heart failure continue or quickly recur if digitalis is then stopped. In other words, with the exceptions noted, the administration of the drug should be continued until decompensation is restored.

Coupled rhythm — an occasional result of digitalis medication in auricular fibrillation — is usually regarded as a definite warning to stop the drug. In order to decide this question for myself, I kept several patients under the effects of the drug for a varying length of time after coupled rhythm had been induced. Most of them were elderly individuals with cardiosclerosis. In each case, digitalis in moderate doses was continued for several weeks after the beginning of coupling. The patients were allowed to walk about. They all felt quite comfortable, there were no ill effects. Improvement was particularly noticeable in one individual who was under treatment for a second severe break in compensation. Besides cardiosclerosis, he had had emphysema with profuse expectoration for years. The typical gross pulse irregularity of auricular fibrillation was present. Digitalis produced coupled rhythm within five days. Medication was continued for two weeks longer although the ventricular rate became as low as 40 per minute. Cyanosis disappeared, expectoration ceased; the patient stated that he had not been so free from cough and expectoration for years. Digitalis was then discontinued; coupled rhythm and improvement remained; the patient passed from observation a few weeks later.

With reference to auricular fibrillation occurring in cardiac decompensation, it remains to be added that the patient must be kept under the influence of digitalis **for months or years** in order to retain proper control of ventricular action and pulse rate. In this manner, individuals can be kept in a good or fair state of compensation for many years. Properly supervised, this long-continued medication can be carried out with no harmful after-effects.

In decompensated cases with rhythmical pulse and **moderate tachycardia**, the latter is often reduced to normal rapidity with gradual restoration of compensation, by the administration of digitalis. This is sometimes brought about by the inception of such digitalis arrhythmias as sinus slowing, sino-auricular block, and blocked auricular beats. As

already indicated, a moderately slow pulse rate is in itself no contra-indication to the further administration of the drug, although in the type under discussion it is apt to be coincident with the full therapeutic effect.

Older patients with **cardiosclerosis** and decompensation usually require larger and longer-continued digitalis dosage than younger individuals with cardiac failure. The former are often comfortable with ventricular rates between 45 and 50; if digitalis is then stopped, edema of the lungs occasionally develops. Such elderly individuals should be kept under full digitalis effects by giving them several courses of the medication over a series of weeks. This is best accomplished by giving one ounce of the tincture (about one drachm daily) for the first week; it is then discontinued for the following week. In the third week about one half ounce of the tincture will be required. It should then be again discontinued for a week. In the succeeding weeks somewhat smaller doses are adequate. In this manner, the heart can be safely saturated with digitalis and the patient kept under its influence for a long time.

Much has been written about the **cumulative effect** of digitalis and the dangers therefrom. Variability in gastric absorption may be one of its main causes. Unless the usual mild toxic symptoms are meant (headache, nausea, arrhythmias), digitalis cumulation is a very infrequent phenomenon, and its dangers have undoubtedly been exaggerated. Digitalis has been administered in innumerable instances for weeks and months, and even years, with no dangerous effects. In only one instance did I observe symptoms which might be interpreted as due to a cumulative effect. A woman of 40 with a double mitral lesion, orthopnea, anasarca, cyanosis, and typical auricular fibrillation entered the hospital *in extremis*. Infusion of digitalis in one half ounce doses was given every four hours. After three ounces had been taken the patient's condition was improved. Suddenly vomiting, a sudden drop of ventricular rate to 60, and cold face and extremities developed. Hypodermic injections of strychnine and caffeine had no effect upon this condition. Despite these apparently alarming symptoms, the patient felt quite comfortable, orthopnea temporarily disappeared, cyanosis decreased, and the urinary output markedly increased. The patient died two days later from cardiac failure.

The **duration of the action** of digitalis after its discontinuance is not definitely known. From electrocardiographic changes sometimes produced by digitalis (Chapter VI), it would seem that after full therapeutic doses have been given, the effects are continued for some weeks. This substantiates the clinical observation that after thorough digitalization, the effects of the drug are maintained by giving much smaller doses.

It is conceivable that there is one **possible danger** in administering digitalis in unnecessarily large doses over long periods; *i.e.* the produc-

tion of **ventricular fibrillation**, an arrhythmia, which, so far as we now know, is universally fatal. We possess neither experimental nor clinical grounds for this assumption. I predicate it upon the power of digitalis to incite all types of cardiac irregularities, and upon the occasional clinical observation that patients who were apparently improving under full digitalis dosage sometimes die quite suddenly, and no reason for the sudden fatal termination can be found at autopsy. Some of these deaths have been assumed to be due to ventricular fibrillation.

The Use of Digitalis in Valvular Lesions. — Dogmatic distinctions were formerly made regarding the various types of valvular lesions in which digitalis was indicated or contraindicated. Abundant clinical experience has shown that, although not followed by equally good results, digitalis may be given in all forms of cardiac failure, regardless of the valve affected. In general, in rhythmically beating hearts digitalis is not followed by the same rapidly beneficial effects as in auricular fibrillation. Its chief value in the former lies in the reduction of the cardiac rate, in lengthening the diastole, and in strengthening ventricular contractions. In aortic lesions with decomposition and left ventricular hypertrophy, digitalis is not as beneficial as in mitral lesions. This may depend upon several factors: except in the final stages of decompensation, there is less systemic congestion and edema; the cardiac rate is very susceptible to neurogenic and other influences which cause tachycardia. In mitral lesions, tachycardia is not as common, congestion and edema more common, and these are the conditions which are more readily influenced by digitalis. Finally, digitalis may not exert its full action upon the fibers of extremely hypertrophied muscle.

The value of digitalis in **cardiosclerosis with hypertension** and **coronary disease** has often been questioned; indeed, until recently, hypertension was regarded by many as a distinct contraindication to digitalis. Careful clinical study has shown, however, that digitalis in therapeutic doses does not regularly raise the blood pressure; when occasionally increased, the increase bears no relation to the time of administration or dosage of the drug. I have treated patients with hypertensive disease, many of whom had a systolic blood pressure of over 200 mm. of mercury, and I have never observed any pressure increment which could in any way be correlated with digitalis administration. In fact, in those in whom cyanosis and dyspnoea were the chief manifestations, and in whom digitalis gave relief, the blood pressure was often lowered. This may be because cyanosis itself acts as an excitant to the vasomotor center and thus raises blood pressure (Chapter XIX). This disturbing factor is eliminated by the beneficial effect of digitalis upon the general circulation.

Another important consideration in digitalis therapy is the question of its use in **endocarditis** and **pericarditis** during the acute stages. When the heart rate is increased as the result of these acute inflammatory

processes, digitalis has no effect in diminishing the pulse rate. This is also true of other febrile diseases, such as pneumonia, grippe, etc. Hence the use of digitalis for the persistent tachycardia of inflammatory conditions is very apt to be followed by disappointing results.

In this connection, I wish to point out a reason for the futility of digitalis in **chronic pericarditis** with dense and extensive adhesions. The latter act mechanically, like a vise, hindering normal systole and diastole. I observed one instance in an adult who had rheumatic arthritis and irregular temperatures for several weeks. Auricular fibrillation was present. The obscure physical signs — a scarcely audible or palpable apex beat — pointed to myocarditis as the probable cardiac lesion. A roentgenogram of the chest showed a somewhat enlarged cardiac shadow. Digitalis was given for several weeks with no effect upon the slowly increasing heart failure or upon the fibrillation. At necropsy, the valves were normal, the myocardium not extensively diseased, there was a tightly adherent pericardium that encircled and fixed the entire heart. In another instance, a case of polyserositis of unknown origin in a young male with dyspnoea and cyanosis, X-ray plates showed definite, sharply defined calcareous plaques in the pericardium. Auricular fibrillation with a fairly regular and slow ventricular rate was present. The cardiac impulse and sounds were weak. Here, also, it seemed probable that the vise-like action of an adherent and thickened pericardium was an important etiological factor in the cardiac failure and cyanosis. Digitalis had no influence upon the symptoms or arrhythmia.

Speaking broadly, in **decompensated valvular and cardiovascular disease** in which infection is either quiescent or non-existent, I have found the following classification of value from the viewpoint of the probable effect of digitalis medication:

1. **Mitral lesions with rhythmic hearts:** Improvement only slowly or sometimes not at all upon digitalis. Rest is apparently the largest factor in the improvement.

2. **Mitral lesions with auricular fibrillation:** Unless decompensation is extreme or long continued, improvement very rapid under digitalis. The irregular cardiac activity is quickly controlled, and, with it, decompensation is usually promptly relieved.

3. **Aortic lesions with slight or moderate ventricular hypertrophy:** The drug is not of much value, rest is the important factor.

4. **Aortic lesions with extreme ventricular hypertrophy:** Reaction to digitalis not good, possibly because there is not sufficient healthy cardiac muscle upon which the drug can act. Even if auricular fibrillation is present, digitalis is not apt to be followed by beneficial results.

5. **Cardiosclerosis with decompensation:** Those with cardiac failure and edema, with or without hypertension, are apt to react well to the Karrell diet (*q.v.*), and digitalis and theobromine sodium salicylate administered on alternate days. In cardiosclerosis and hypertension

with uremia and no edema, digitalis has very little effect in the relief of decompensation.

Summing up the effects of digitalis medication, the following **conclusions** are warranted:

1. The best single criterion of the amount and duration of digitalis administration is its clinical effect.
2. The beneficial effect of digitalis depends chiefly upon producing increased contractile power of the cardiac musculature.
3. The **gradual** production of arrhythmias is usually coincident with full clinical effects.
4. If decompensation demands the continuance of digitalis, the **rapid** onset of arrhythmias does not contraindicate its further use.
5. Atropine sulphate, grain $\frac{1}{160}$ to $\frac{1}{80}$, three times a day, given internally or subcutaneously at the beginning of medication, occasionally prevents nausea and vomiting in susceptible individuals.
6. The hypertension of cardiovascular disease does not contraindicate the use of digitalis.
7. To derive clinical benefits quickly, digitalis should be given in full therapeutic doses.
8. When digitalis acts promptly in small doses, the effect is likely to be temporary. For permanent improvement, long-continued medication is usually required.
9. Digitalis occasionally produces epigastric sensitiveness and hunger pangs, which appear to be due to heightened vagal tone. These symptoms precede vomiting by one or two days, and indicate a full therapeutic digitalis effect.

TINCTURE STROPHANTHUS

This possesses no advantages over the tincture of digitalis. Its disadvantage is that it varies considerably in strength. It should be given in one half or one drachm doses, and not in the smaller quantities usually recommended.

Crystalline strophanthin (Thoms) is a most powerful glucoside. The best preparation is that put up in ampoules containing 1 c.c. of a 1 per cent solution. The dose is from 10 to 15 minims; this should be slowly injected, preferably intravenously. It is of remarkable efficacy, particularly in the acute heart failure of auricular fibrillation. Within a very short time, sometimes after one hour, the injection may be followed by slower and more regular ventricular activity, with a correspondingly good effect upon dyspnoea and cyanosis; frustrate beats may quickly disappear, edema show some evidence of clearing up, and diuresis increase. If the urgency of the case demands it, the injection should be repeated at the end of 24 hours. In other words, these injections can in a remarkably short time produce the effect which only occurs after several days of the usual method of digitalis

administration. On the other hand, the effect is more evanescent; hence, in the decompensation of cardiac disease, it is necessary to follow up these injections by digitalis given in the usual manner. Strophanthin is also indicated in acute cardiac failure from cardiorenal disease, especially if pulmonary edema is present. There is one **caution** necessary respecting strophanthin; namely, it is **contraindicated**, or should be used very sparingly if the patient is already digitalized, otherwise very serious toxic symptoms may immediately follow. For example, in a patient with mitral regurgitation, extreme decompensation, and rhythmical pulse, who had long been under the full effects of digitalis, this precaution was not heeded; an injection of 1 c.c. of strophanthin was immediately followed by auricular fibrillation, a condition which lasted until death a few days later and undoubtedly hastened the fatal termination.

TINCTURE OF SQUILLS

In doses of 15 to 30 minims this has an action on the circulation similar to digitalis. Like the latter, it may produce sinus arrhythmia and heart block, apparently by increased vagal inhibition. It possesses no advantages over digitalis, and is, in addition, irregular and uncertain in action. Squills is often prescribed in pill form as a diuretic.

APOCYNUM

Apocynum or Canadian Hemp, given in fluid or solid extract, has been studied to a slight extent; it has no regular or marked effect upon the circulation.

CAFFEINE AND ITS DERIVATIVES

Caffeine produces vaso-constriction by its action upon the vasomotor center; it also has a slight effect upon the heart itself. Its main therapeutic value rests upon the action on the kidneys. Since cardiovascular disease involves the kidneys, a discussion of caffeine and its derivatives is of importance.

Caffeine acts primarily as a diuretic, thereby increasing the output of urine. The manner of its action is still in dispute; some ascribe it to an irritating effect upon the renal epithelium, others to an effect upon the renal circulation. Caffeine increases principally the watery constituent of the urine; there is also an increased output of solids, especially of sodium chloride, and, to a lesser degree, of the nitrogenous constituents.

The best method of caffeine administration is in the form of caffeine sodium salicylate, a readily soluble salt. A 20 per cent solution of this salt in distilled water lends itself admirably to hypodermic administration; the dose is from 15 to 30 minims.

Theobromine Sodium Salicylate. — This salt produces more marked diuresis than caffeine. Another advantage is that it has no effect upon

the vasomotor center, and hence does not produce blood pressure changes. The drug is administered in 7 to 15 grain (0.5 to 1 gm.) doses three or four times daily, well diluted in water. Because of an unpleasant taste, it can be given in wafers. If the medication upsets the stomach, it may be given in smaller doses more frequently. It can also be administered per rectum by the Murphy drip method. Finally, it can be given intravenously according to a method which I have developed.

This **intravenous method** seems of importance because the drug is particularly indicated in uremic conditions with vomiting, delirium, and disturbances of the sensorium, in which theobromine cannot be given by mouth, or if so given, the amount absorbed is very slight or nil. At first I employed a solution of ten grains dissolved in 200 c.c. of water, once or twice a day. This method was discarded because it was too cumbersome and did not permit sufficiently frequent administration. Now I use 5 per cent solutions, the percentage ordinarily used in animal experimentation. Pharmacological examinations show that the specific gravity of a 5 per cent solution at 20° C. is 1.0228, and its alkalinity equivalent to that of a 2.4 per cent solution of sodium bicarbonate. Sodium bicarbonate in this concentration, or even stronger, is sometimes given intravenously in diabetic coma. A 5 per cent theobromine sodium salicylate solution heated in a closed vessel at 96° C. for one hour, again for one hour, and then for one half hour, shows slight yellowish discoloration and a very slight loss of alkalinity, but remains perfectly clear. Sterilization does not alter the solution. The dose I usually employ is 15 grains, *i.e.* 20 c.c. of the 5 per cent solution. In one instance 30 grains (40 c.c. of the solution) were given in a single dose. The stock solution should be resterilized by boiling immediately preceding injections. If the entire solution enters the vein, there is no local reaction. If a few drops find their way into the subcutaneous tissue, some induration, ecchymosis, or slight pain lasting a few days sometimes follows. In one instance, through a misunderstanding by one of the house staff, the solution was injected subdermally; a local skin slough and ulcer resulted. The injections are never followed by systemic reactions. The solution is readily prepared and sterilized. While 20 c.c. of a 5 per cent solution have been found a convenient standard, the dosage can be modified to suit individual requirements. The ordinary dosage can be given intravenously daily for several days.

It has been shown that the diuretic function of the kidney becomes fatigued by long-continued theobromine administration, hence it is advisable to give the drug in the customary oral method in courses of two or three days with intermissions of similar periods. Theobromine sodium salicylate (diuretin) is particularly indicated in the dropsy of cardiorenal disease. Given on alternate days with digitalis, it often enhances the action of the latter, not only in cardiorenal but also in purely valvular disease. As an adjuvant, theobromine is often combined

with the Karrell diet. Because of its importance, this diet and the method of its administration require detailed description.

The **Karrell diet** consists in a daily diet of 800 to 1000 c.c. of milk; it is preferably given in glassfuls of six ounces every three hours. This is usually termed a "Karrell day." On Karrell days the patient should be in bed or at rest. The milk should be sipped slowly. Patients rarely complain of thirst, but occasionally become so hungry that they suffer from hunger pangs and faintness. Under these conditions, I allow them dry toast, crackers, perhaps also an apple or orange in amounts just sufficient to curb extreme hunger. An excellent routine procedure in moderately severe edema of cardiorenal origin is Karrell diet and theobromine sodium salicylate for two days, alternating with digitalis and an appropriate renal diet (*q.v.*) with somewhat restricted fluid intake for the succeeding two days. In most cases I believe it advisable to continue theobromine therapy and Karrell diet, at intervals, long after edema has disappeared and the patient feels well. These intervals should be gradually lengthened; for example, Karrell diet and theobromine may be given for a day, at first twice weekly, then once weekly, then once monthly. In this manner it seems possible to retain and continue for months, and occasionally for years, the improvement gained by the initial, more intense therapy. In some instances of cardiorenal disease with extreme anasarca, the intensive treatment outlined — digitalis, theobromine, and Karrell diet — yields remarkable results: losses of from 10 to 15 pounds during the first week, and from 20 to 40 pounds in several weeks, are not exceptional. Coincident with reduction of edema, other serious symptoms such as semistupor, dyspnoea, cyanosis, and manifestations of hypertension, often improve or disappear. Beneficial results derived from this therapy are most marked in those in whom myocardial insufficiency is a prominent clinical feature.

As a **modification** of the **Karrell diet** and as a method of promoting diuresis in patients in whom the urinary output is approximately normal in amount, I have restricted the fluid intake to only 500 c.c. daily. The liquids allowed are water, sweetened or unsweetened lemonade, and coffee or tea with very little milk. When necessary, only sufficient solids of the kind already mentioned are added to control severe hunger. Patients rarely complain of thirst; when present, it is partially relieved by small quantities of cracked ice, or by rinsing the mouth with water. I give this restricted and modified Karrell diet for two days, with theobromine sodium salicylate in 7 to 15 grain doses to the amount of 45 grains daily. Then follow two days of digitalis with appropriate cardiorenal diet and somewhat restricted fluid intake. By this method, I have occasionally achieved results which did not follow the ordinary Karrell diet. Older patients with long-standing edema frequently voided from 60 to 80 ounces, and occasionally 100 ounces, on the day succeeding the second theobromine day, the usual time of

most marked diuretic action. Coincidentally, edemas quickly cleared up, and cyanosis and especially dyspnoea very rapidly and often remarkably improved. These secondary results may have been due not only to the elimination of water, but also to elimination of toxic substances.

STRYCHNINE

In animal experiments, strychnine has been shown to have a vaso-constrictor influence, and hence has been assumed to be of value in low blood pressure and shock. However, careful observations of its effect on the circulation in health and in cardiac disease have shown no influence upon the urinary output, edema, blood pressure, or heart rate,—the usual criteria of any change in the circulation. The claim, occasionally made, that in some intangible way strychnine acts as a circulatory 'tonic' by its effect upon the nervous system is not substantiated by any clinical evidence.

NITRITES

This is the vasodilator group. It consists mainly of nitroglycerin, nitrite of potassium and sodium, amyl nitrite, erythrol tetranitrate, and mannitol hexanitrate. Amyl nitrite is the emergency drug of this class and is considered in detail in another section (Chapter XXI). Nitroglycerin, the drug most often prescribed, acts as a peripheral arteriolar dilator. The usual dose is $\frac{1}{100}$ grain three times daily. It may be given hypodermically or by mouth. It is chiefly used in circulatory disease for lowering blood pressure in hypertension and for the relief of precordial pains. It is believed that the latter is accomplished by the relief of spasm of the coronary arteries. In some cases of hypertension I have administered as much as $\frac{1}{10}$ of a grain of nitroglycerin subcutaneously three times daily with little or no effect upon hypertension or precordial symptoms. These and other inconstant results following nitroglycerin may be accounted for by the varying amounts of arterial thickening and consequent variations in the dilating power of the arterioles. In edema of the lungs with hypertension and nephritis, nitroglycerin in large doses (from $\frac{1}{8}$ to $\frac{1}{10}$ gr. hypodermically), combined with the usual cardiac remedies, occasionally relieves the overburdened heart by its effect upon the peripheral circulation.

Nitroglycerin is of most value in cardiosclerosis with moderate hypertension and labile vasomotor mechanism (Chapter XXI). Its administration may then result in fairly long-continued lowered blood pressure and in relief of symptoms. If the use of nitroglycerin be limited to such cases, it becomes a very valuable drug. If, however, it is used in continued hypertension with advanced coronary disease, nitroglycerin is apt to be followed by disappointing results for reasons already mentioned. Erythrol tetranitrate and mannitol hexanitrate have a slower and more continued action than the others of the vasodilator group.

CAMPHOR

This drug, especially in the form of camphor in oil, given hypodermically, has been a favorite circulatory remedy for many years. Careful clinical observation, however, has shown that it has no demonstrable influence on the circulation; *i.e.* it exerts no influence on diuresis, blood pressure, or pulse rate. In animal experimentation there is a slight effect upon the pulse pressure. The use of spirits of camphor in depression of the nervous system, as, for example, in fainting spells, is sometimes of value because of its reflex action.

ALCOHOL

Discussion of alcohol is here limited to its effect upon the circulation. In animals, alcohol slightly increases cardiac contractility. In human beings, when given in large quantities, it produces a marked fall of blood pressure by its action upon the vaso-constrictor center and upon the heart muscle. Small quantities of alcoholic beverages seem occasionally to augment cardiac contractility in decompensated cardiac disease, but the effect is slight and inconstant, and its value problematical.

ACONITE

Recent investigations have shown that the strength of the tincture of aconite, the preparation usually used, varies considerably, and that, even when properly standardized, the dose ordinarily administered is much too small. Aconite does not slow the heart rate in cardiac disease. Its presumed therapeutic value is based upon experimental investigation in animals in which cardiac rate, contractility, and blood pressure are lowered as the result of a central vagus effect. When given in very large doses, the drug sometimes increases ventricular irritability. This has been shown by the production of extrasystoles by pressure over the pneumogastriacs of patients who had been given the drug.

SPARTEINE

This drug is the alkaloid of the common broom. Formerly it was used as a cardiac tonic because it was presumed to have an action like digitalis. When injected into animals, it slows the pulse and slightly raises the blood pressure. Clinically, these effects have not been observed; however, it must be added that careful investigation of its action is lacking. The dose advised ranges from $\frac{1}{2}$ to 5 grains; probably 2 grains of sparteine sulphate is a safe dose for an adult.

SUPRARENAL EXTRACT — ADRENALIN

Its vaso-constrictor action, especially when given intravenously, is well known. It may be used in circulatory collapse accompanied by a sharp fall of the systolic blood pressure, or in hypertension with rapidly falling pressure, the latter indicating myocardial insufficiency. Injections of the drug in these conditions are sometimes followed

by decided though temporary improvement. Adrenalin has also been advised in so-called constitutional hypotension (Chapter XIX), a condition in which cardiac failure is absent. The main symptoms in these individuals are those referable to vasomotor disturbances. The hypotension itself rarely requires medication, but the accompanying symptoms may be relieved by adrenalin. The best method of administration is the standard sterile solution in the strength of 1 to 1000; the dose is from 15 to 30 minims given internally, or, preferably, hypodermically or intravenously.

MORPHINE

This drug produces slowing of the heart rate by its effect upon the medullary center. Injections in dogs are sometimes followed by complete heart block. In man, morphine is a cardiac 'stimulant' only in the sense that, by its effect upon the central nervous system, the patients become less restive and irritable. Thus regarded, it is often an excellent aid to other drugs in such acute circulatory conditions as paroxysmal tachycardia, acute onset of auricular fibrillation, and sudden cardiac failure with dyspnoea.

BROMIDES

These are indicated in tachycardia from any cause, or in those in whom extrasystoles cause subjective sensations. In the former, where morphine may also be indicated, the bromides enhance and prolong the morphine effect.

Other sedatives which may be employed to combat restlessness and sleeplessness are chloral hydrate and veronal; they may be given alone or combined with small doses of morphine or codeine.

ACETATE OF POTASH AND SODA

Their diuretic action is usually slight and depends upon the non-metallic salt — the acetate — for its effect. It is occasionally of advantage to alternate these drugs with the more active diuretic, theobromine sodium salicylate. The dose is from 15 to 30 grains, given in solution every two or three hours.

SALINE CATHARTICS

Brisk catharsis is sometimes of value in starting diuresis. The best saline for this purpose is the sulphate of magnesia.

CALOMEL

The drug is sometimes of benefit in cardiac dropsy; it is of less value in edema of renal origin. The dose generally recommended is 3 grains three times daily for two to four days; after an interval of a few days,

unless some contraindication exists, it may be repeated in the same dosage. Its action is ascribed to direct stimulation of the renal epithelium.

VENESECTION

This procedure has its sphere in plethoric cases with cyanosis and dyspnoea. It possesses a double advantage: temporary decrease of heart work by decreasing blood volume, and removal of toxic products. The amount to be withdrawn must be individualized; it should be at least 250 c.c.

AUTOGENOUS AND STOCK VACCINES, AND SENSITIZED SERA

I have employed autogenous vaccines in a number of cases of endocarditis due to the streptococcus viridans, but have not been able to discern any benefit from their use. There was no effect upon the temperature or toxic symptoms, nor was there any change in the clinical course of the disease. In a few cases that came to necropsy in which this therapy was employed, nothing was found to indicate that the vegetative process on the valves was checked in the slightest degree.

I have also administered stock vaccines in a small number of cases of endocarditis which seemed clinically of streptococcic origin but in whom the organism could not be isolated from the blood. Though I observed no ill effects from the injections, they had not the slightest therapeutic result.

In a number of instances of streptococcus viridans infection, I employed serum derived from horses sensitized to this coccus. The effect of such injections was also entirely negative.

COLLOIDAL SILVER PREPARATIONS

Recently, interest in the use of silver salts in septic conditions has been revived by the administration of various colloidal silver salts in the treatment of acute rheumatic and bacterial endocarditis. I have observed a few cases in which they were injected intravenously. In one, a streptococcus viridans infection, 20 injections were given in two or three day intervals without benefit; the patient died with the symptoms of cerebral embolism. In another instance, a child with acute rheumatic endocarditis, a few injections were followed by a rather sharp fall in temperature. In the same ward, I had under observation at this time a male adult of 20 who suffered from acute rheumatism and from acute endocarditis and pericarditis with effusion. The cardiac involvement ran a very stormy course: the temperature reached 105° and remained between 104° and 105° for one week; dyspnoea, chiefly due to the very large amount of pericardial exudate, was extreme. The patient received neither silver injections nor serum therapy of any kind. After one week the temperature fell suddenly, signs of pericardial effusion quickly disappeared, the patient rapidly

convalesced with the physical signs of a permanent aortic lesion. Thus are contrasted two cases of an acute endocardial infection both of whom rapidly convalesced, one with, the other without, any attempt at specific therapy. The inference is clear that there is no proof in the first case that the injections caused a recession of the acute endocarditis. In the enthusiastic reports regarding the use of colloidal silver in endocarditis, I have found no instance of 'cure' which could not be explained upon the basis of an acute endocarditis which had quickly run its febrile course, or upon the basis of a longer duration with the usual remissions. In other words, I do not believe that the intravenous use of silver preparations is warranted in any type of endocardial infection.

MASSAGE — PASSIVE MOTION

As a result of careful observation of many cases of cardiac disease, it is my impression that patients are allowed in bed or at absolute rest too long after compensation has been restored, and after the inflammatory process has run its course. Two to three weeks after the quiescence of inflammatory symptoms and after restoration of compensation constitute, I believe, the average time for complete rest. After that period, the judicious use of massage, active and passive motion, and exercise aid the circulation and produce a more rapid return to normal circulatory conditions. No form of exercise or massage should be pushed far enough to cause dyspnoea, distress, rapid heart action, or unpleasant subjective symptoms. Massage and passive motion have also some value as added therapeutic aids in long-continued cardiac decompensation in bedridden patients. The chief contraindication in such individuals is extreme continued dyspnoea. In such cases, massage should be limited to the extremities and to the muscles of the back; the abdomen and anterior part of the chest should not be included. When edema and venous stasis are present, it may be of advantage to stroke the limbs in the direction of the venous and lymphatic current, *i.e.* from the periphery to the trunk. This sometimes decreases the local engorgement.

The kind of massage to be employed in cases of restored compensation must be individualized. Usually light superficial massage alone is tolerated. Deep vigorous massage and kneading of the muscles commonly produces discomfort and pain, and, by exciting the patient, may cause rapid heart action and dyspnoea. The amount of passive motion must be similarly studied in every individual case. Flexion and extension of the smaller joints — the fingers, wrists, ankles, and toes — should at first be practiced gently once daily; later, the larger joints (knees, elbows, hips, and shoulders) should be flexed and extended. If these procedures are not followed by pain, cyanosis, tachycardia, exhaustion, or dyspnoea, more vigorous and oft-repeated passive motion

can be employed. Massage and passive motion do not necessarily require the services of a trained attendant; in any case, however, they should at the outset be carried out under the direct supervision of the medical attendant, so that he can carefully observe any possible ill effects following their use.

Calisthenics — Medical Gymnastics — Resistance Exercises.—

These are occasionally indicated while the patient is still in bed. They require the more active coöperation of the patient and make more demands upon his circulatory reserve. Rapid breathing, dyspnœa, increased and prolonged pulse rapidity, or subjective feelings of faintness and exhaustion are the best clinical guides that gymnastics or exercise have exceeded safe limits and have caused marked diminution of the cardiac reserve power. It should be emphasized that these treatments are only added therapeutic aids, and cannot replace circulatory drugs when indicated. The use of dumb-bells and of carefully graduated exercises as measured by the ergostat are also of value. According to some observers, delayed return to the normal blood pressure following dumb-bell exercise constitutes a good reaction, and is to be regarded as evidence of good cardiac reserve power.

Walking is the best, simplest, and most accessible of all the forms of more active exercise. Some years ago, a system of carefully planned and graduated hill climbing with interspersed resting benches was popular on the continent (Oertel treatment), but arbitrary insistence upon fixed distances to be covered or hills to be climbed leaves out of consideration the only safe and cardinal guide of circulatory endurance; namely, the patient's cardiac reserve power as measured by his subjective sensations, a factor which varies daily or even hourly. On the other hand, walking at a moderate pace on the level or up a slight incline for a distance or length of time well within the patient's circulatory power is a much safer and more elastic rule, and approaches more nearly the normal demands and mode of life that the individual will later follow.

Hydrotherapy.— The usual hydrotherapeutic procedures consist in tepid baths, sponging with tepid water followed by a quick cool or cold sponge over the entire or upper parts of the body, tepid or cool shower baths, needle baths, or sprays of water of varying temperatures applied to various portions of the body. Except for very warm baths, after the patient is properly inured to them, the above measures are applicable to any type of cardiac disease in which compensation has been well established. These measures have their especial application, however, in a large group of patients who, in addition to their organic disease, suffer from **marked neuropathic tendencies**. Such individuals are apt to have vasomotor instability, such as hot or cold extremities, frequent flushes, etc. They may suffer from dyspnœa of purely subjective origin not correlated with exercise or with any discernible cardiac change. They complain of indefinite pains in the chest, neck, or arms.

They frequently suffer from headache and anorexia. In other words, this type of patient, well compensated, suffers from that train of cardiac symptoms found in the so-called cardiopathic individuals, in whom there is no organic disease. Doubtless the knowledge of the presence of organic trouble plays a rôle in unduly directing the patient's attention to otherwise trivial symptoms. To some extent, also, I have found that this neuropathic tendency has been caused by consultation with physicians who have exaggerated the importance of the physical ailment and have not sufficiently relieved the mental disquietude and fear of sudden death which exists in many patients with cardiac disease. Naturally, where pathological changes are present, it will require careful discrimination between the so-called neurotic symptoms and those emanating from the organic lesion. In those in whom the diagnosis of an added neurotic ailment has been established, the hydrotherapeutic measures already outlined exercise a very favorable influence. Regarding the special procedure to be employed in individual instances, it is necessary to remember that in no case should early treatments be so brusque or rigorous as to frighten or in any way upset the patient. Tepid baths or sponges should be employed at first; later, more vigorous measures—cool sprays and showers—may be used. In this manner, the neurotic symptoms, which are very real and often disheartening to the patient, may gradually disappear.

Carbon Dioxide and Oxygen Baths.—In a different therapeutic category belong those baths in which the water is naturally or artificially charged with carbon dioxide or oxygen. The gas bubbles surround the body of the patient, produce a sensation of warmth, and cause dilatation of the superficial vessels. The claim is made that by the use of these gas-impregnated baths, congestion is relieved, the pulse rate slowed, dyspnœa decreased, and the overburdened heart correspondingly benefited. There are conflicting reports regarding the effect of these baths upon blood pressure; it is raised according to some, lowered according to others. In those I have observed, there was no constant rise or fall of the systolic blood pressure following the baths. There seems little room for doubt, that, when taken at Spas and health resorts, gas-impregnated baths are of benefit; this is not, however, because of their direct effect upon the circulation, but because of the difference in environment and enforced rest from business and worry, and because of the more moderate diet and more regular and physiological mode of living. The baths act upon the circulation in the same manner as a mild stimulating exercise, and, in my opinion, upon this alone rests their efficacy upon the circulation. Their use should therefore be limited to well-compensated patients or to those with mild disturbances of compensation. Even here, the baths should not be prescribed universally, but only to those in whom observation has shown a good reaction during and following the bath. Cold extremities, a sense of exhaustion, rapid breathing, accelerated pulse rate, dyspnœa, constitute distinct

contraindications. I regard the possible effect upon the blood pressure as of less importance.

The usual **routine** of a series of **carbonated baths** given at **Nauheim** consists of a first immersion lasting from five to seven minutes in a bath of the temperature of 95° F. The temperature of the succeeding baths is gradually reduced to about 86° F. and the immersion time slowly increased to 20 minutes. The bath is omitted every third or fourth day. A full course consists of two series of 20 baths each, with an interval of three or four weeks. Oxygen and 'Nauheim' carbonated brine baths can be prepared at home by dissolving salts producing oxygen and carbon dioxide gas respectively, in a proper quantity of water. Naturally the greatest disadvantage of such home baths is that there is no change of environment, with all its attendant and important beneficial features. However, in the group of cardiac patients already described, who suffer largely from neurotic in addition to organic complaints, gas-impregnated baths, even when taken at home, may be followed by excellent results.

Types of Patients Suitable for Spa Treatment.—The question frequently and insistently arises in private practice as to the class of cardiac patients who should be sent to the continent for 'Nauheim' treatment. Nauheim is mentioned because it is the most popular and best known among the patients themselves. Until some American health resort becomes as popular and can offer the same or similar well-balanced facilities for restful environment and medical supervision, the question must be fairly met and intelligently considered. The answer must depend, not upon the type of valvular lesion or cardiovascular disease, but chiefly upon the state of cardiac compensation. Patients severely decompensated, for example those suffering from marked edema, cyanosis, and dyspnoea, should be advised against taking a trip abroad for Spa treatment. They are more benefited by proper treatment at home. There are some patients, however, otherwise intelligent, who will go to almost untold risks and expense to achieve what they conceive to be a life-saving cure, and this directly against medical advice. I have observed several such patients who returned home worse than when they left; very few were improved. One of those not benefited was a physician with marked cardiosclerosis, edema, and cyanosis, who left for Europe against advice; he returned home a few months later *in extremis*. The patients I have found most improved were those who, besides mild decompensation, were nervous, high-strung, or worried, and who at the foreign Spas were able not only to have enforced rest and quiet, but also to quickly divorce their minds and thoughts from domestic and business cares. It is to individuals of this type that Spa treatment should be recommended with the great probability that it will be followed by good results.

Tonsillectomy is discussed in this connection because it has assumed much importance since the demonstration that the tonsils are often

the portals of entry for endocardial infections. Tonsils which are palpably diseased or which become frequently infected, should be properly and thoroughly removed if the general condition of the patient warrants it. This statement also applies to adenoids. However, I hold conservative views regarding the removal of apparently healthy tonsils with the idea of preventing the further spread of endocarditis. I believe that when valvular disease is already present, the removal of small and healthy looking tonsils does not tend to prevent reinfection. This view is not invalidated by the fact that tonsils which look normal in the throat show, upon removal, various pathological changes, both macro- and microscopically. Because of their function as filtering agents and constant contact with, and exposure to, bacteria, the tonsils can scarcely ever represent normal lymphoid structures. It is still a moot question whether recrudescences of endocarditis have been prevented by the routine and radical practice of tonsillectomy in all children with valvular lesions. Thus, in one excellent report, such tonsillectomies did not affect the frequency of recurrences, nor the course of the disease.

Extraction of Teeth. — As the result of careful and long-continued observation, I believe that endocarditis in itself does not warrant more radical or careful dentistry than is usually required for diseased teeth and purulent foci in other otherwise normal individuals. Dentistry here as elsewhere should follow sane lines. As with tonsillectomy, I regard routine extraction for the prevention of endocarditis as uncalled for (Chapter XI). Extensive extractions upon the supposition that exceedingly small pus foci frequently produce endocarditis is in my opinion unwarranted by general clinical experience and by the negative results following such practice in patients with endocarditis.

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CHAPTER XVII

DIET, AND RENAL AND BLOOD TESTS IN CARDIAC DISEASE

FROM the dietetic standpoint, and because of the frequent correlation of cardiac with renal disease, it is necessary to make a clinical distinction between purely cardiac cases with no or very slight secondary renal involvement, and those in whom renal involvement is the primary or predominant feature. It is also necessary, in both types, to remember that we are dealing with chronic conditions, and that the dietetic régime will have to be carried out for months and years. Hence the diet should not be unnecessarily harsh or restricted, and consideration should be given, as far as possible, to individual idiosyncrasies and tastes.

In patients with **compensated valvular disease** with or without hypertrophy, without renal involvement or hypertension, it is not necessary to make any radical change from the usual dietary of balanced rations of an individual in health. There is no necessity for strict observance of any special diet, nor for the limitation of fluids. On the other hand, there should be sensible restriction about excesses of any kind of food or drink.

The Use of Beverages. — To patients who have been accustomed to small amounts of alcohol in the form of beer or light wines, I have been in the habit of allowing a slight amount of these beverages, provided observation shows no deleterious effects upon the circulation as noted by the pulse rate and blood pressure. I have usually limited the amount to one glass of beer or to a small glass of light wine taken at the principal meal. I have observed such patients for years, and I have found no ill effects upon the cardiac condition ascribable to this practice. Similarly, if patients have been in the habit of taking tea or coffee without any ill effects, a cup of weak tea or coffee may be allowed daily. In obese individuals, the carbohydrate and fat intake should be somewhat restricted in order to produce some loss in weight; but this should be a gradual process and so fitted to individual needs that the diet does not become irksome, and hence apt to be given up. As substitutes for carbohydrates and fats, cabbage, spinach, lettuce, beans, carrots, and mushrooms may be suggested.

Those with **myocardial insufficiency and dyspnoea** without edema are usually too ill to relish a normal amount of solid food, so that the patient's own limitations and dietetic inclinations are usually sufficiently safe guides. But in view of the possibility of visceral stasis and of edema, the fluid intake should be somewhat restricted; about 500 c.c. in addition to solid and semisolid nourishment is a safe limit. In cases of myocardial insufficiency with edema, pleuritic transudates, or marked visceral congestion, a strict Karrell diet for several successive days or in courses of several days each, as already outlined in the discussion of theobromine (Chapter XVI), is an excellent aid to drug therapeutics. The Karrell diet reduces the salt intake to a minimum. Even after disappearance of edema, and when comparison of the salt intake with its output in the urine proves a normal relationship and no chloride retention, only a moderate amount of salt should be allowed, in order to overcome any tendency to edema by salt retention.

The importance of diet in those cardiorenal cases in which the kidneys especially require attention requires a brief account of some of the **modern methods of kidney study** and diagnosis, and their correlation with diet and therapy. Besides the routine examination of the urine for normal and abnormal chemical and microscopical constituents, other methods have come into use to estimate renal function and efficiency by the power of elimination of certain substances or foods taken internally or given subcutaneously. The status and value of some of these tests and their correlation with clinical data have not yet been definitely fixed. Two of these tests consist in the study of iodide of potash excretion, and of the elimination of lactose injected intravenously. Both of these have been practically discarded. The first test requires a long time for its completion and gives inaccurate results; the lactose test is too cumbersome and is likewise inaccurate. Probably the most important method at the present time consists in the estimation of the excretion percentage in the urine of a dye which is injected subcutaneously. Phenolsulphophthalein is usually chosen for this study. Briefly, the method employed is as follows:—

Twenty minutes to one-half hour before the injection, the patient is given 200 to 400 c.c. of water. The patient is then catheterized, the time noted, and the catheter kept in place. One c.c. of a carefully prepared sterile solution, containing 6 m.g. of the dye, is injected intramuscularly in the lumbar region. The urine is allowed to drain into a test tube which contains a drop of a 25 per cent sodium hydroxide solution, and the time of the appearance of the first pinkish tinge is noted. The catheter is then withdrawn and the patient is asked to void into two different receptacles at the end of one and two hours respectively. Sufficient 25 per cent sodium hydroxide solution is added to make the urine decidedly alkaline. This causes a yellowish or orange color in acid urines which suddenly changes to a brilliant purplish

red when the urine becomes alkaline. Sufficient water is now added to each specimen to make 1000 c.c. The solution is mixed; a small portion is then filtered and compared with the standard color. In those patients seen in office consultation, where the phthalein test seems of value, I have employed the following routine: The patient is asked to void; he is then given a glass of water and the dye is injected. He is sent home and is asked to void his urine two hours after the injection; this specimen is then examined for the percentage of dye excretion. I have found this method sufficiently exact for ordinary purposes; it obviates the necessity for catheterization, and requires very little time.

As colorimeters the Dunning, Hellige, or Duboscq instruments may be used; or one can manufacture as I have done a practical and sufficiently accurate colorimeter by using a series of bottles or tubes which contain the solution of phenolsulphophthalein in multiples of 5 per cent; the tubes are sealed and their respective percentage labeled; they may then be used as color standards. If the dye first appears in the urine within 15 minutes of the time of its injection, and 40 to 50 per cent is excreted within two hours, the test is regarded as normal. For clinical purposes, the two-hour excretion test alone is sufficient; this saves catheterization of the patient. In both renal congestion secondary to circulatory disturbances and in contracted kidney, the percentage and output in two hours is usually decreased. It requires the clinical examination to determine which of the two factors — renal congestion or primary nephritis — is the cause of the decreased phthalein output.

For the purpose of testing quantitatively for urea, salt, and water output in the urine, a balanced diet containing a measured amount of solids (starches, sugar, fats, proteids, and salts) and of fluids (milk, water, tea) is given. Schlayer's diet or some modification may be employed. An excellent and well-balanced ration is that prescribed by Mosenthal for testing the renal function. This is as follows: —

NEPHRITIC TEST DIET

All food is to be salt free.

Salt for each meal is furnished in weighed amounts.¹

All food or fluid not taken must be weighed or measured after meals, and charted.

Allow no food or fluid of any kind except at meal times.

Note any mishaps or irregularities that occur in giving the diet or collecting the specimens.

¹ One capsule of salt, containing 2.3 gm. of sodium chloride, is furnished for each meal. The salt which is not consumed is returned to the laboratory, where it is weighed, and the actual amount of salt taken is calculated.

BREAKFAST, 8 A.M.

Boiled oatmeal, 100 gm.
 Sugar, 1-2 teaspoonful
 Milk, 30 c.c.
 Two slices bread (30 gm. each)
 Butter, 20 gm.
 Coffee, 160 c.c.
 Sugar, 1 teaspoonful } 200 c.c.
 Milk, 40 c.c.
 Milk, 200 c.c.
 Water, 200 c.c.

DINNER, 12 NOON.

Meat Soup, 180 c.c.
 Beefsteak, 100 gm.
 Potato (baked, mashed, or boiled), 130 gm.
 Green vegetables, as desired
 Two slices bread (30 gm. each)
 Butter, 20 gm.
 Tea, 180 c.c.
 Sugar, 1 teaspoonful } 200 c.c.
 Milk, 20 c.c.
 Water, 250 c.c.
 Pudding (tapioca or rice,) 110 gm.

SUPPER, 5 P.M.

Two eggs, cooked in any style
 Two slices bread (30 gm. each)
 Butter, 20 gm.
 Tea, 180 c.c.
 Sugar, 1 teaspoonful } 200 c.c.
 Milk, 20 c.c.
 Fruit (stewed or fresh), 1 portion
 Water, 300 c.c.

8 A.M. — No food or fluid is to be given during the night or until 8 o'clock the next morning (after voiding), when the regular diet is resumed.

Patient is to empty bladder at 8 A.M. and at the end of each period, as indicated below. The specimens are to be collected for the following periods in properly labeled bottles:

8 A.M.—10 A.M.; 10 A.M.—12 N.; 12 N.—2 P.M.; 2 P.M.—4 P.M.; 4 P.M.—6 P.M.; 6 P.M.—8 P.M.; 8 P.M.—8 A.M.

Outside of hospitals, as in sanatoria or at home, a similar dietetic régime can be carried out if, instead of quantities measured in grams and cubic centimeters, their known equivalents in cupfuls, glasses, and teaspoonfuls be substituted. This is a modification that I have carried out where careful weighing of the various food constituents could not be done, and I have found the results sufficiently accurate for clinical purposes.

The amounts, specific gravity, and sodium chloride content, and the urea determination by the hypobromite method or preferably the nitrogen content by the Kjeldahl method are separately determined for each two-hourly specimen, and also for the night specimen. In other words, there are six determinations for the day, and one for the night specimen. In this manner, besides other data, a comparison of the amounts and specific gravity of the day and night urines is made. If the specific gravity is low and shows only slight variations in the different specimens, and the night urine is larger in amount than that voided during the day, this speaks for the presence of a chronic interstitial nephritis.

Because of evaporation by the lung and elimination of water by the skin and intestines, the amount of urine is normally less than the fluid intake. Approximately all sodium chloride, and about 90 per cent of the nitrogen ingested, is excreted by the urine. Thus, the chemical urine tests above indicated give valuable information regarding the retention of salt and nitrogenous products in the body.

In order to determine which of these chemical tests is of most clinical value, I have added in a small series of cases a study of the non-protein nitrogen in the blood, the estimation of carbon dioxide tension of the expired air by means of the Fredericia apparatus, and the phthalein output; I have also observed the alkali tolerance of the urine by giving the patient measured amounts of bicarbonate of soda until the urine became alkaline. I have not yet arrived at any definite conclusions. However, I believe that a very careful study of the clinical phenomena, of the amount and concentration of the night urine, and of the ordinary examination of several specimens of urine taken within 24 hours, will allow us to judge with sufficient clinical accuracy the amount and type of the retained excrementitious products and even of the degree of renal insufficiency in the majority of cases. In other words, clinicians may soon hope to reap the advantages of the careful scientific work of the laboratory by comparison of its results with ordinary bedside methods.

Chemical examination of the blood has assumed great importance, especially since simpler and accurate methods have replaced the former cumbersome and less accurate determinations. The important constituents sought for are non-protein nitrogen, creatinine, and uric acid. The knowledge thus gained is not yet fixed or positive, but there seems to be a correlation between an abnormal amount of non-protein nitrogen retained in the blood, a decreased phthalein output, and clinical evidence of a severe nephritis of predominant vascular type. Not only has our diagnostic and prognostic knowledge been enhanced by these blood examinations, but the dietary of nephritis has assumed a more rational and scientific aspect. The subject of salt restriction has already been mentioned. In nephritics with abnormally increased amounts of non-protein nitrogen in the blood, a low protein and high carbohydrate diet is indicated. The amount of fluid should be restricted if urine measurements show decreased elimination of water. If the amount of urinary excretion is normal, it is advisable to occasionally allow large quantities of fluids — as much as several liters within 24 hours — in the attempt to flush the system and thus get rid of toxic products.

In **nephritis with excessive and abnormal amounts of non-protein nitrogen** in the blood, attempts have been made to therapeutically attack the disease upon a chemical basis. Sugar solutions have been administered intravenously or by the Murphy drip. In cases of lessened blood alkalinity (so-called acidosis), I have in several cases given intravenous injections of 500 c.c. of a 5 per cent solution of bicarbonate of

soda. One instance was that of a patient of 64 with cardio-nephritis, regular pulse, hypertension, general anasarca, and nocturnal dyspnoea, who had been ill over one year. Therapy consisting in digitalis, theobromine, appropriate diet, and occasional Karrell days over a period of several weeks, had only a very slight influence upon the symptoms and course of the disease. 500 c.c. of blood were withdrawn by venesection and the same amount of a 5 per cent bicarbonate solution injected. Within 24 hours improvement began and continued uninterruptedly. At the end of two weeks, edema and dyspnoea had entirely disappeared. In another case, a man with cardiovascular disease, a systolic blood pressure of 250, aortitis, very marked left ventricular hypertrophy, and attacks of nocturnal dyspnoea relieved only by morphine, a similar procedure was employed. The injection was followed by improvement for several days. In less urgent cases I have tried venesection combined with a 10 per cent bicarbonate solution per rectum given by the Murphy drip. Possibly a simple bedside method like the use of the Fridericia apparatus for the determination of CO_2 content in the alveolar air may give sufficiently accurate estimations parallel to the chemical examination of the blood for acidosis, and thus be of advantage in determining the cases suitable for alkaline therapy.

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CHAPTER XVIII

MANAGEMENT OF CARDIAC DISEASE—MARRIAGE IN WOMEN WITH VALVULAR DISEASE

ASIDE from therapy and diet, other questions regarding the management of compensated and decompensated cases of cardio-vascular disease arise. Some of the commoner of these are: Shall a patient return to work? What type of work shall he follow? Shall medication be continued and, if so, how long? How shall reinfection be prevented? Shall women with heart disease be allowed to marry?

With respect to these questions, no matter what the type of lesion, there are **two preliminary fundamental considerations** to be determined, namely: the **degree of compensation** and the **state of quiescence** of the disease. With quiescent compensated lesions, valvular or myocardial in nature, the main restriction regarding exercise should be the kind, rather than the amount, provided always it be well within the patient's cardiac reserve power. This statement requires some modification, for the type of cardiac disease plays a rôle which requires some individual discrimination. For example, patients with tremendous hypertrophic left ventricles from aortic valvular lesions are scarcely able to maintain long-continued effort without soon encroaching upon their cardiac reserve. In general, however, it may be stated that even quiescent compensated cases should avoid all exercises which call for sudden or sharp exertion, as swimming, running, and tennis playing. On the other hand, golf is an excellent form of moderate exercise. It entails the necessity of being away from business and out in the open for a number of hours, in themselves very desirable. In exercise as well as in work it should be emphasized that patients should keep well within their individual limits of fatigue.

The question of **occupation and vocation** for patients with cardiac disease has recently received wide consideration from the lay as well as from the medical standpoints. It is gradually being recognized that many individuals with cardiovascular disease are not thereby necessarily precluded from attempting to earn a livelihood, and that, if proper work be chosen, they may become self-sustaining members of the community. Occupations and vocations at which patients sit or stand

are preferable to those which require walking or stair climbing. Positions in counting houses, clerical work, draftsmanship, light manufacturing industries, working at lathes or small machinery, watchmen, are examples of the work which these patients may safely follow; but just because of these sedentary and easy occupations, exercise out in the open, chiefly walking, should be advised. It is, I believe, a therapeutic error to attempt to avoid all circulatory strain by having these patients pass an almost muscularly inert existence, for it is only by mildly stimulating the circulation by appropriate gentle exercise that the heart and circulatory apparatus are kept at their proper individual level of efficiency. In this respect the heart does not differ from other weakened muscles whose strength is enhanced by moderate, well-planned, and individualized exercise. Mild, appropriate dumb-bell exercises and other calisthenics should be advised when walking is not feasible, or as additions to the latter.

In this connection, a brief account of **functional efficiency tests** for estimating the cardiac reserve power is appropriate. Various tests have been proposed to estimate the muscular efficiency and working power of the heart. A fluoroscopic examination is one means which has been employed. If orthodiascopic observation reveals an increase in the size of the cardiac outline after exercise (*i.e.* if the heart dilates instead of contracting), it is regarded as a sign of an inefficient heart; while if contraction follows exercise, the heart is regarded as efficient. Differences in systolic blood pressure following exercise, and differences in pulse pressure in the standing and sitting positions, are also employed as guides of cardiac sufficiency. An attempt has likewise been made to estimate the muscular efficiency and cardiac load by a standard quotient obtained by dividing the pulse pressure by the systolic pressure. Another formula consists in estimating the heart load as the resultant of the division of the pulse pressure by the diastolic; this should normally approximate 50 per cent. Still another test for functional efficiency consists in the observation of the time required for the blood pressure to return to its level following exercise. It has been found that, in normal individuals, the systolic blood pressure is raised immediately after a moderate amount of work. With more severe exercise, the maximum systolic pressure is not reached until one or more minutes after exercise had ceased. This observation has recently been applied in a study of the functional capacity of diseased hearts; instead of an ergometer the foot pounds of exercise or work are computed by the use of dumb-bells and bars of various weights; these are lowered and raised a certain number of times to known heights.

Objection to Functional Efficiency Tests. — It appears to me that there are several objections to the various methods of using blood pressure readings as tests for cardiac efficiency. Blood pressure depends upon important factors other than systolic output (Chapter XIX); the cardiac rate in circulatory failure often increases dispro-

portionately to pulse pressure; increased cardiac work does not necessarily imply increased systolic output or increased blood pressure; the state of venous distention in the splanchnic area — an unknown factor because it cannot be measured — may considerably influence and vitiate the calculations. Finally, the various estimations can only give temporary information regarding the circulation at the time the tests are made. Far simpler, more readily applicable, and apparently as accurate as the above tests, is the usual clinical method of observing the patient during various forms of exercise — standing, walking, bending, etc., — and of estimating the cardiac reserve by noting the rapidity of pulse and respiration, dyspnoea, and such subjective sensations as discomfort and exhaustion. Information derived from the patient's subjective sensations is of special importance, and cannot of course be calculated by any instrumental method. The above routine can be further amplified in appropriate cases by allowing the patient to follow his ordinary occupation, if not too laborious, for part of or the entire day, and then noting the effect upon his circulation and cardiac reserve. Such data, in addition to a very careful clinical examination, are, I believe, preferable to mathematical "efficiency tests."

When, in cardiac disease, compensation has long since been established, drugs of the digitalis group are not indicated. It is, however, very important to decide whether a patient who suffers only occasionally from cardiac failure should continue medication after compensation has been restored. This has already been partially discussed (Chapter XVI). When signs of cardiac failure occur readily after moderate exercise, patients should be continuously under the effect of some digitalis preparation. This is especially true of the cardiac failure accompanying auricular fibrillation; prolonged compensation can be best maintained by administering small doses of digitalis for stated periods; for example, for one week and then discontinuing the drug for a similar or longer period. Thereafter smaller doses may be administered in a similar manner. In patients with rhythmically beating hearts, in whom tachycardia is one of the signs of decompensation, the bromides may be of value in addition to digitalis.

MARRIAGE OF WOMEN WITH VALVULAR DISEASE

This question is one which requires careful consideration. The possible dangers arising from marriage are twofold: the strain and excitement incidental to **coitus** and that from **pregnancy**. Regarding the former, I have seen several instances of hemoptyses immediately following coitus in patients with auricular fibrillation, and in one with cardiosclerosis and hypertension. I have also observed several in which intercourse was followed by violent tachycardia. With one exception,

the cardiac condition of these patients would have been considered fairly satisfactory under usual conditions.

The marriage question cannot always be settled upon the grounds of health alone, although that is naturally the phase of importance to the physician. Some writers maintain that patients with heart disease should never marry, no matter what the type of lesion. I believe that this view is extreme. One frequently encounters mothers of large families who doubtless have had valvular disease for many years, and who have gone safely and normally through pregnancy and parturition. Many of these patients were never aware of their disease; in some the lesion was discovered in the course of a routine examination.

Marriage should naturally not be considered at the time when the patient shows the slightest degree of decompensation. In those who have only recently recovered from heart failure, it is a safe rule to interdict marriage until at least two years have passed without further break in compensation. An exception is noted later in reference to aortic lesions and extreme cardiac hypertrophy. The interval mentioned — two years — is of course purely arbitrary, but seems to agree best with clinical experience. The same time should be set regarding the interval of freedom from inflammation in valvular lesions. If, for example, there have been rheumatic endocarditic recrudescences, as shown by louder murmurs, slight febrile attacks, pericarditis, tachycardia, arrhythmias, or other clinical manifestations, marriage should not be advised, for the chances are that another outbreak will occur before one year has passed. In short, marriage (and pregnancy) may be considered safe if excellent compensation and freedom from endocardial exacerbations persist during the two-year period. The physician who has not had the opportunity to observe the patient within the prescribed antenuptial period must necessarily be guided by the history and the physical signs.

Of the two factors, decompensation and quiescence of the lesion, I believe that the latter is the more important. In my experience, more danger and more fatalities have resulted in subsequent pregnancies from marriage occurring when lesions were active than from mild decompensation. Pregnancy seems to light up dormant or only partially active cardiac processes. Cardiac symptoms often begin early, sometimes in the third month of pregnancy. At the beginning, simple or paroxysmal tachycardia may be present. Slight fever may appear; when due to endocardial exacerbations, its presence is of serious import. The occurrence of new or louder valvular murmurs, or of fresh pericarditis, may also furnish direct evidence of inflammatory recrudescence. Hemoptysis is not infrequent. As pregnancy advances, unless the above or similar manifestations recede or are checked, dyspnoea and cyanosis gradually supervene and, with them, the usual symptomatology of frank cardiac decompensation: edema, orthopnoea, enlarged liver, pulmonary congestion, etc. The greater circulatory

demands made by the growing placental and foetal circulation also play a rôle, in all probability. Labor, induced or spontaneous, does not always terminate the circulatory embarrassment, for insidious endocarditis may continue, and death result from some complication or from circulatory failure.

Fundamental considerations which refer to all types of valvular lesions with reference to marriage have already been mentioned. It now remains to partially differentiate between these upon the basis of clinical experience. I have found that rheumatic **mitral stenotic lesions** are the most dangerous to pregnant women. These patients readily develop paroxysmal or, more often, simple tachycardia, which may last during the greater part of pregnancy. The rapid heart action itself may produce such dyspnoea or discomfort that the induction of premature labor is indicated. Hemoptyses are common. Bronchitis with sibilant breathing and mucous râles over the entire chest are not unusual. As occasional complications in the puerperium, embolic infarcts in the lower extremities or in the lungs may be mentioned.

The history of pregnant women with **mitral stenosis** and **auricular fibrillation** presents a varied clinical picture. Some of these patients date their first break of compensation at a first or second pregnancy which had been carried to full term. On the other hand, I have observed cases who went through successive pregnancies with mitral stenosis and auricular fibrillation, with no cardiac complications or symptoms. One of these deserves brief mention: When first examined, she was fifty years old, with general anasarca, orthopnoea, auricular fibrillation, a double mitral lesion, and an old rheumatic history. She had had eighteen children without cardiac symptoms. The latter began only three years after her menopause which commenced five years before. Another patient, fifty-three years of age, was the mother of five children. She had a rheumatic history and a double mitral lesion with auricular fibrillation for many years. During her pregnancies the cardiac symptoms were very slight and of the same nature as those occurring when she was not pregnant; these consisted in occasional dyspnoea and tachycardia.

There seems to be no way of determining in advance the favorable or unfavorable subjects for pregnancy in those with auricular fibrillation, except by the method already outlined. Extreme caution in advising marriage is, of course, necessary because of the known tendency of patients with auricular fibrillation to decompensate.

Patients with simple **mitral regurgitant** lesions are most fit for pregnancy and most apt to go through gestation without untoward cardiac complications. When the latter do occur, they are usually of the mild decompensatory type rather than due to recurrence of endocardial exacerbation.

Pregnant women with **aortic lesions** suffer chiefly from tachycardia. This is true of those with, as well as those without, marked ventricular

hypertrophy. In the latter, however, tachycardial attacks occur more frequently, are more readily invoked, and of longer duration. Decompensation is comparatively rare in those with only moderate or slight hypertrophy; when extreme, cardiac failure is apt to occur early in pregnancy, a tendency increased by the rapid heart action. Such patients should not be permitted to become pregnant even if the lesion is quiescent and compensation in the non-pregnant state is good.

In all types of decompensated endocardial lesions occurring during pregnancy the question of the **induction of abortion** or of **premature labor** arises. Severe cardiac failure in early pregnancy (before the fourth month) or slight decompensation which does not yield to treatment are absolute indications for immediate emptying of the uterus. This indication is not vitiated by the fact that some of these mothers may, by protracted rest and medication, carry the child to viability or even to full term without further complications. The life of the mother is the prime consideration and should not be jeopardized, as it would be, in an attempt to continue the pregnancy when cardiac decompensation is present at an early stage. My observation has been that an abortion, surgically clean and skillfully performed, is only slightly if at all more dangerous in cardiac patients than if performed for other reasons on those with normal hearts. I am also strongly in favor of terminating an early pregnancy if it is evident that there exists a continuance or recrudescence of endocarditis. As already indicated, such evidence may consist in the onset of paroxysmal or constant tachycardia, of extrasystoles or other arrhythmias, of frequent hemoptyses, and of changes in the physical signs.

If signs of decompensation, or of fresh endocarditis, appear between the end of the fourth month and the time of viability (the seventh month), in view of the somewhat more serious operative procedure required to induce miscarriage, the decision regarding either procedure hinges chiefly upon the severity of the cardiac complications. If decompensation is mild or the evidence of fresh endocarditis not severe, appropriate therapy should at first be attempted for about a week or ten days. Should the symptoms then disappear and the patient improve, pregnancy may be allowed to proceed until the period of viability. If decompensation or endocarditis do not react well to therapy or become suddenly severe and threatening, it is much safer to induce miscarriage.

In the interim between the seventh and the ninth month of gestation the decision regarding the interruption of pregnancy in decompensated cases or in those with recrudescing endocardial lesions is not of such vital importance, because the premature induction of labor in proper hands adds scarcely any risks. The question of waiting a month or two until a more natural process of normal labor occurs must depend upon the cardiac condition; that is, if there is any reason to fear the slightest increase of cardiac complications, it is both wiser

and safer to have pregnancy terminated soon than to wait until full term.

Aside from the induction of abortion or of premature labor, the treatment of the various cardiac symptoms occurring during pregnancy is that already described in the chapters on endocarditis, arrhythmias, and cardiac failure.

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CHAPTER XIX

BLOOD PRESSURE

Physiological Considerations. — Blood pressure observation has taken its place as a routine method of examination in clinical medicine. Its value and importance in health and disease are based upon the results of experimental physiology, which demonstrated that various factors are concerned in the estimation of blood pressure. These are: (1) Cardiac Energy; (2) Peripheral Resistance; (3) Elasticity of the Arterial Wall; (4) Volume of the Circulating Blood; (5) Viscosity of the Blood.

1. **Cardiac Energy.** — From the physical standpoint, cardiac work depends upon the amount of and velocity with which the blood is pumped into the arterial system. This in turn largely depends upon the amount of venous blood brought to the heart. If greatly diminished, as, for example, by section of the splanchnics, ventricular filling and, consequently, systolic output are considerably diminished. Other factors being equal, an increased volumetric output raises, a decreased output decreases, the aortal pressure. In the normal animal, the pulse rate is retarded by raising arterial pressure, and accelerated by lowering it. This reaction does not usually occur after vagus section; hence it is probably due to action upon the vagus center, partly reflex and partly direct. In animals, the centripetal nerve to this center is the depressor which sends terminal filaments to the ventricular musculature and probably also to the aorta. If this nerve is cut or stimulated peripherally, it has no effect upon the heart action or blood pressure. If its central end is stimulated, there is a marked fall in blood pressure and heart rate. The depressor thus acts as a defensive mechanism against unduly high blood pressure. Its aortal filaments are stimulated by undue distention of the vessel.

2. **Peripheral Resistance.** — With other factors unchanged, the blood pressure is increased with increased, decreased by lessened peripheral resistance. The tension of the normal artery depends upon its tonus, which in turn chiefly depends upon the vasomotor mechanism. To a lesser extent, this statement applies to some of the veins. Normal tone is governed by a proper balance between the vasodilator and vasoconstrictor mechanisms, as derived from the regulatory center in

the medulla. There are other subsidiary centers in the spinal cord; this is demonstrated by the fact that, after destruction of the bulbar center, the arteries gradually recover their tone. In the experimental animal, general vasomotor tone is readily affected by distant and near reflexes. After stimulation of the depressor nerve the splanchnic vessels become dilated and blood pressure falls, but stimulation of all the other centripetal nerves raises blood pressure. The abdominal vessels, innervated by the splanchnics, have the greatest effect upon the general blood pressure because they can contain a large amount of blood and are easily influenced reflexly. It is important in this connection to emphasize that reflex lowering or raising the blood pressure does not simultaneously affect vascular areas in the same way.

3. **Elasticity of the Arterial Wall.** — Because of their elastic distensibility, a large proportion of the force of ventricular systole is stored up in the larger arteries, which, by stretching and elastic recoil, act as a reservoir of power after systole has ceased. By this means the strain of systole upon the cardiac musculature, as well as upon the arterial wall, is considerably diminished. If the arterial tree represented a rigid system, the systolic blood pressure, and hence the force of the impact, upon the arteries would be greatly increased.

4. **Volume of the Circulating Blood.** — The possible content of the arterial and venous systems is much larger than the actual amount of blood found in the body. The disproportion is equalized by peripheral contraction, hence blood volume has only a very slight effect upon blood pressure.

5. **Viscosity of the Blood.** — It is evident that the degree of viscosity may have some influence upon blood pressure. Up to the present time, however, there are no clinical or experimental data which have any bearing upon the subject.

All these factors are present in the human being as well as in the experimental animal. Their value and importance vary not only relatively but absolutely from time to time, and the blood pressure is the resultant of these variants. Although we possess no methods or instruments by which individual agencies making up the clinical blood pressure can be separately calculated, one should at least attempt to gauge them by careful examination and by such significant data as the thickness of palpable arteries, existence of plethora, etc.

Clinical Estimation of Blood Pressure. — The instruments which are used in the clinical estimation of blood pressure are called sphygmomanometers and are of two main types, the mercurial and the aneroid. Both fundamentally depend upon calculating the power exerted in partial or complete obliteration of an artery in terms of a column of mercury or of an aneroid pressure indicator, respectively. In its clinical application, the brachial is the artery chosen for compression. This is accomplished by means of a standard 12 cm. broad, inflatable cuff

placed around the upper arm. To the cuff are attached two tubes: one connected with the small metal or rubber hand pump for the purpose of inflation, the other connected with the mercurial column or aneroid instrument. A disadvantage of the latter is that the spring may lose its resiliency and hence the instrument become inaccurate. If standardized sufficiently often by comparison with a mercurial sphygmomanometer, the aneroid manometer is as useful for clinical purposes as the mercurial instrument. Its advantages are that it is made compactly, so as to be more readily carried about, and the dial can be read more conveniently than the millimeter markings in a column of mercury.

Arm cuffs are of two types: one, stiff, made of leather; the other, soft and made of cloth or silk. Though either can be used, I prefer the soft cuff because it is pliable and more readily and quickly placed in position.

Blood pressure estimations of the brachial artery should be taken at **the level of the heart**. For this purpose, the elbow of the patient is placed comfortably upon a desk or table. The cuff is then snugly applied to the bare arm. The arm should be relaxed, otherwise muscular tonicity tends to produce an incorrectly high blood pressure reading. Edema also causes abnormally high blood pressure, because much of the pressure within the cuff is used up in displacing the edema.

The introduction and use of the sphygmomanometer have shown that the estimation of blood pressure by radial palpation alone is erroneous and untrustworthy. There are various methods of determining the systolic blood pressure by manometric readings; these are (1) the palpatory, (2) the visual, (3) the graphic, (4) the auscultatory.

According to (1) the **palpatory** method, the systolic blood pressure is that degree of cuff pressure exerted on the brachial at which the radial pulse becomes no longer palpable. (2) The **visual** method consists in compressing the brachial beyond the point of obliteration, and then gradually lowering the pressure by cuff deflation until the first mercurial oscillation becomes visible; this point marks the systolic blood pressure. In the (3) **graphic** method, the mercurial manometer has an additional connection with a rubber bulb inclosed in a hermetically sealed small glass globe; the latter is attached to a recording tambour, so that differences, as shown by the mercurial oscillations, are transmitted to the tambour and recorded on a revolving drum.

(4) The **auscultatory** method is the one usually employed because of its accuracy. The cuff is applied and inflated until the brachial is obliterated, that is, beyond the point at which mercurial oscillations are visible. The stethoscope is then applied to the brachial at the bend of the elbow, and the cuff is gradually deflated until sharp distinct taps are heard; this marks the systolic blood pressure. In typical instances, it is possible to separate the auscultatory phenomena into **five distinct "phases."** The **first** is the one just described and, as noted, establishes

the **systolic pressure**. The artery beneath the cuff being empty, the first pulse wave produces sudden arterial tension with resultant short, popping sounds. With a gradual drop in blood pressure by continued deflation, the sharp tap gives way to a murmur, somewhat resembling a superficial, rough pericardial friction sound. This marks the **second** phase. It is probably caused by sufficient blood passing under the cuff to produce swirling arterial currents which mask the click of the first phase. By further deflation and consequent lessened pressure on the brachial, this murmur ceases and the **third** phase begins. It is marked at the beginning by short tapping sounds which, with continued deflation, gradually change to muffled taps; this is usually regarded as the beginning of the **fourth** phase, and as marking the **diastolic pressure**. Some dispute exists regarding the etiology of the sounds in the fourth phase. It seems probable that, with the gradual approach of diastolic pressure, a steady arterial stream begins to flow beneath the cuff between the arterial pulsations; this acts as a kind of buffer and thus produces the muffled and dull sounds characteristic of the fourth phase. The dull note is finally extinguished by a constantly increasing column of blood in the artery, until all sounds cease. This marks the **fifth** or last auscultatory phase. The usual difference between the fourth and fifth phases is from 5 to 8 mm. of mercury.

The various phases as outlined are not always well defined or distinguishable. There are several **variations** from the normal. If the brachial artery is small, the separate phases may be indistinct or inaudible. Again, the first phase may not be represented by a sharp click, but by a murmur. Occasionally, the second phase is entirely absent, especially in aortic disease with high systolic blood pressure. Such differences probably depend upon the varying strength of arterial eddies interfering with the usual sharp taps of sudden arterial distention.

The **maximal normal blood pressure** is still a matter of dispute. In a statistical table by Fisher representing 19,339 accepted candidates for life insurance, including ages from 15 to 60, the average systolic pressure was 129 mm. of mercury. As shown by these and other statistics and observations, it appears that 150 mm. marks the extreme normal systolic blood pressure of a healthy **middle-aged** individual. The normal **diastolic** pressure in adults is from 70 to 90 mm. of mercury. There is a gradual rise in systolic and diastolic blood pressure with advancing age.

There are certain **physiological variations** which must be borne in mind when taking blood pressure estimations. The systolic blood pressure is increased by expiration; in forced expiration this may amount to 5 to 10 mm. There is also a slight diurnal variation accounted for by differences in physical and psychical states of the individual at various times of the day. The blood pressure is lowest during the first part of natural sleep. The ingestion of meals has a varying in-

fluence upon blood pressure; this depends upon different degrees of vasodilation of the abdominal vessels with compensatory superficial vasoconstriction.

There are certain factors which require brief comment because of their influence upon blood pressure. Moderate amounts of alcohol do not regularly raise the blood pressure in man. In the experimental animal, large doses produce a fall in blood pressure, due to an effect upon the vasoconstrictors and upon the heart. When smoking tobacco, there is a temporary rise of blood pressure from stimulation of the peripheral and central vasoconstrictor mechanism. Excitement—pleasurable or otherwise—sometimes produces a temporary rise of blood pressure. Exercise and muscular exertion have a similar effect; in young healthy individuals the blood pressure soon drops to its usual level; in the middle-aged and old, the rise of blood pressure represents a more marked reaction and lasts longer. Cyanosis may in itself be a cause of abnormally high blood pressure; hence therapeutic measures, like venesection and digitalis (which tend to diminish cyanosis), may directly decrease existing hypertension. This is an important consideration in the treatment of decompensation and hypertension in which cyanosis forms an important element. Another factor in the production of hypertension in some cases of arterial disease is that due to hypertonus; this possible cause for error, however, can be controlled and obviated by repeated compression of the artery with the cuff.

In general, frequent blood pressure estimations are necessary in order to arrive at the correct blood pressure of the individual, and to obviate some of the disturbing physiological and other factors that have been mentioned.

For the purpose of studying **abnormal blood pressure in organic cardiovascular disease**, I have found the classification I devised, embracing the vast majority of cases, to be of clinical value. Despite occasional overlapping, the predominant lesion or type is usually readily recognized.

- I. { (a) Hypertensive cardiovascular disease with myocarditis.
 (b) Hypertension and myocardial insufficiency with labile vasomotor mechanism.
 (c) Uremia.
- II. Myocardial disease and insufficiency without hypertension.
- III. Valvular disease and myocardial insufficiency with and without hypertension.
- IV. Senile and premature arteriosclerosis.

I (a). **Hypertensive cardiovascular disease with myocarditis** includes some of the fairly well-defined groups of the older writers; for example, Huchard's presclerosis, Gull and Sutton's arteriocapillary fibrosis, von Basch's angiosclerosis. It is becoming increasingly evident that the pathological basis of hypertensive cardiovascular disease lies chiefly in disease affecting the arterioles of the heart, kidneys, and

brain. As concomitant changes in the heart, there may be cardiac hypertrophy (mainly of the left ventricle) and moderate or advanced aortitis. The systolic blood pressure commonly varies from 180 to 230 mm. of mercury, the average being 190 to 200. It is important to note that there may exist no parallelism between the degree of hypertension and the extent of kidney involvement, and that hypertension alone need not necessarily be of symptomatic or prognostic importance. The average diastolic pressure of this group is about 100 mm.; if renal involvement predominates, diastolic pressures of 120 or over are found.

I (b). **Hypertension and Myocardial Insufficiency with Labile Vasomotor Mechanism.** — This group consists principally of patients past middle life with only moderate hypertension and with normal or moderately elevated diastolic pressure. The chief characteristic of the blood pressure is its marked daily variation, being as much as 30 mm. of mercury. The symptoms referable to myocardial insufficiency are mild. The patients are usually stout or obese men; the prominent physical signs in the chest are those of bronchitis and emphysema. The palpable arteries are not thickened, and nephritic symptoms not marked despite the presence of a slight amount of albumin and of a few casts in the urine. Pretibial edema is absent or only very slight.

I (c). **Uremia.** — The cardinal symptoms in patients of this group are headache, nausea, vomiting, varying grades of pallor, attacks of paroxysmal dyspnoea, precordial distress, and nocturnal polyuria. There are retinal changes of various degrees. The phenolsulphophthalein test (Chapter XVII) shows diminished output in two hours, the average being between 15 and 30 per cent. As a result of renal test meals containing weighed amounts of water, salt, carbohydrate, and nitrogenous constituents (Chapter XVII), and of chemical examination of the blood, we find as a rule low, fixed specific gravity for the day and night urines, decreased elimination of salt, water, and urea in the urine, and abnormal amounts of non-protein nitrogen and sometimes of uric acid in the blood. When uremic dyspnoea is marked, there is evidence of diminished blood alkalinity (so-called acidosis). This may be roughly estimated by determining the amount of bicarbonate of soda, administered internally, required to render the urine alkaline. Other more refined and direct methods consist in determining the carbon dioxide content of the alveolar air by the Priestly-Haldane bag or by the Frederica apparatus, or by testing the blood chemically according to the Van Slyke method. It has recently been emphasized that the **differentiation** between the "cardiac" and "cerebral" types of hypertension can be made by careful observation of the diastolic pressure, which presumably serves as the better index of the peripheral resistance. The diastolic pressure is between 120 and 140 in the "cerebral" cases, *i.e.* in those who suffer from such typical uremic signs as headache, vomiting, and retinal changes; it is considerably less in those in whom "cardiac" manifestations are especially prominent.

II. Myocardial Disease and Insufficiency without Hypertension. — Patients in whom myocarditis is the predominant pathological condition show little or no hypertension. The diastolic pressure is sometimes quite low, so that, even with a normal systolic, there is an increase in the pulse pressure and hence in the cardiac load.

III. Valvular Disease with Myocardial Insufficiency with and without Hypertension. — The valvular lesions in which the systolic blood pressure is high (without evidence of general arterial disease) are rheumatic affections of the aorta, especially aortic regurgitation. In this, the blood pressure may be between 180 and 200 mm.; the diastolic pressure is abnormally low, the average being 40 to 25; it is occasionally zero. There thus exists a very marked cardiac overload. It has been shown that, in this valvular lesion, the systolic blood pressure in the femoral is often much higher than in the brachial. This difference I have found to be as high as 50 mm. of mercury in individual instances. The normal difference between leg and arm blood pressures is from 5 to 10 mm., hence the diagnostic importance of measuring the arm and leg blood pressure in cases of suspected aortic regurgitation in whom such usual clinical signs as typical murmurs and the Corrigan pulse are absent.

Unless arterial disease or cyanosis is present, mitral lesions are unaccompanied by hypertension. With beginning heart failure, the systolic blood pressure may become subnormal. In combined valvular disease of the mitral and aortic valves, blood pressure depends upon the clinically predominant lesion. For example, if aortic regurgitation is the more prominent, the blood pressure will be characteristic of that lesion; if the mitral predominates, the pressure will be normal.

IV. Senile and Premature Arteriosclerosis. — Under this caption are grouped those cases with tortuous and thickened visible arteries. The heart valves and aorta present various degrees of intimal thickening and lime deposits; the main coronaries and their branches are thickened, there is marked myo-fibrosis, the heart may be small or only moderately enlarged. Such patients often have only slight hypertension or even normal blood pressure, unless renal involvement is clinically marked.

In addition to the foregoing classified groups of organic cardiovascular disease, several other important conditions which have a direct bearing upon blood pressure estimations require brief description.

Exophthalmic Goiter. — A characteristic of this disease is the variability of the blood pressure readings; there are marked differences from day to day. When the disease is of moderate severity, hypertension is the rule, the range being between 160 and 180 mm. of mercury. Extreme hypertension without accompanying cardiovascular disease is rare.

Lead Poisoning. — During attacks of lead colic there is usually a rise of the systolic blood pressure. This is attributable partly to the

pain present during the attack, but chiefly to the spasm of the peripheral arterioles. Plumbism which causes disease of the kidneys and arteries, and ends in nephritis and arteriosclerosis naturally produces hypertension because of the presence of these lesions.

Increased Intracranial Tension. — Hemorrhage, meningitis, and brain tumors are the usual causes of this condition. As a consequence of increased intracranial tension, cerebral anemia results, and with it, an increase — sometimes marked — of the systolic blood pressure. In addition to the hypertension, other manifestations of cerebral anemia may consist in headache, vomiting, vertigo, choked disk, and true bradycardia.

Cyanosis is so frequently found in broken compensation, that its effect in producing hypertension requires special consideration. Its action may be ascribed to an effect, in a milder degree, similar to that of the blood in asphyxia. In the latter direct stimulation of the vasomotor center is assumed. Cyanosis in itself may frequently account for hypertension in decompensation from any cause, for, with decrease of cyanosis, the blood pressure often returns to the normal and remains there. Therefore, by treating cyanosis, diverse therapeutic measures such as digitalis medication and venesection may have a direct and beneficial effect upon hypertension.

Functional Hypertension, Hyperpiesis. — Besides hypertension due to known pathological change or abnormal clinical condition, there remains a small group of patients in whom at present no cause for hypertension can be found. This has been termed hyperpiesis. In this group may be mentioned females approaching the menopause and a few sufferers from gastric disturbances of a neurotic nature. The following is a typical case:

Female, age 55, menopause 8 years ago. Some years previously she had gastric symptoms: belching, dizziness, and hunger pains. Her present complaints have lasted several months; these are dizziness, slight nausea, and at times belching. Dyspnoea, edema, or urinary changes are absent; the sodium chloride, water, urea excretion, and sulphophthalein output are normal; the Wassermann blood reaction is negative. The amount of non-protein nitrogen in the blood is also normal. Physical examination reveals no evidence of cardiovascular disease. Despite all these findings the systolic blood pressure is regularly about 200, the diastolic normal. It is possible that such cases are referable to extreme susceptibility of the vasoconstrictor center to reflex influences. Until more of these cases have been studied by means of chemical examination of the blood and by functional urinary tests, and have been followed to necropsy, they must for the present be provisionally regarded as of functional, non-organic origin.

Hypotension. — This term applies to a group of adults presenting no evidence of organic disease and in whom the systolic blood pressure

is about 100 in males, 90 in females. Some of these individuals are of robust physique. They never suffer from decompensation. They often run a symptomless course and are then only accidentally discovered in the course of routine clinical examinations. If symptoms be present, they consist of rapid fatigue following moderate exertion; the vasomotor system is unstable and susceptible to nervous influences of various kinds. The patients flush or become pale readily. They are excitable, often become dizzy or complain of feeling faint; indeed, their symptoms overlap to a great extent those described under "Weak Heart" (Chapter XX).

BLOOD PRESSURE IN CARDIAC ARRHYTHMIAS

It is sometimes important to estimate blood pressure when various types of pulse irregularities are present. The methods to be employed in the commoner of these require brief description.

Sinus Arrhythmia and Heart Block. — Since the beats at the wrist are of equal force, only the routine methods are required for the blood pressure determination.

Alternation. — There is a rhythmic sequence of stronger with weaker beats. The systolic blood pressure of the stronger contractions can be estimated in the usual manner. Their diastolic pressure can thus also be estimated if it be higher than the systolic of the smaller beats. This fact is readily determined during the course of the examination by palpating the radial; the number of radial beats which come through will then be just half those at the cardiac apex.

Coupled Rhythm. — The tension of the initial beats of the couplets in auricular fibrillation with coupled rhythm is not identical, but the difference is usually slight, so that for our purpose the coupling of auricular fibrillation may be grouped with that occurring in extrasystoles. The systolic blood pressure of the initial stronger beats is calculated in the usual fashion. If the diastolic pressure of these is greater than the systolic pressure of the weaker contractions of the couplet, then again the usual routine in measuring the diastolic pressure of the stronger is followed.

When **extrasystoles** occur frequently, and at irregular intervals, it is necessary to use a special method similar to that for auricular fibrillation (*q.v.*) in order to estimate the systolic blood pressure.

In **auricular fibrillation** with gross irregularity in the force of the beats, the ordinary routine cannot be applied, for it can only estimate the systolic blood pressure of the strongest, and the diastolic of the weakest beat at the moment that the blood pressure is taken. When all ventricular contractions are propagated as pulse waves, and the radial beats on palpation feel equal in force, my observations have shown that the pressures of the individual beats do not usually vary more than 10 mm. of mercury; hence the ordinary method of

estimating the systolic levels are sufficiently accurate for clinical purposes. With extreme irregularity in the strength of the radial, and with many frustrate and abortive beats, various methods have been devised to arrive at an approximation of the actual blood pressure. According to one method, that of James and Hart, the cuff and stethoscope are first applied in the usual manner. These observers have found that, although the rate be irregular, the number of ventricular contractions for each minute is approximately the same. Their procedure is as follows: the number of radial beats which pass under the cuff, between pressures of 150 and 140 mm. of mercury, for example, are counted, as well as the corresponding number of ventricular contractions occurring during the same time. The difference between these represents the number of frustrate or abortive beats. Similar observations are then made for systolic blood pressures between 140 and 130, 130 and 120, etc., until the systolic blood pressure of all the beats which pass under the cuff have been taken. The number of palpable radial beats found at the various blood pressures is multiplied by the highest systolic limit set for that group; these products are added and then divided by the corresponding heart rates at the apex. The result gives the "average" systolic blood pressure. One objection to the method is that the "average" includes many abortive beats (called by the authors pulse deficit) which have no effect upon the circulation since they propel no blood through the arteries. A better and more correct plan is to "average" the effective beats only, *i.e.* those that actually have some effect upon the circulation by the production of pulse waves. This is attempted in the "fractional" method of Kilgore, according to which the diastolic pressures are also calculated. The systolic and diastolic pressures are plotted on a chart, the number of beats occurring at the various systolic and diastolic pressures being marked by points; these are then connected so as to form a smooth graph. In this manner it is possible to make fairly exact estimations of the blood pressure in auricular fibrillation.

THERAPEUTICS OF HYPERTENSION

This subject has been discussed in part in a previous chapter (Chapter XVI). Before remedial measures are considered, it is well to emphasize that hypertension is almost always a conservative or compensatory process. As a corollary, it follows that hypertension itself does not necessarily require medication, for it is usually but an index of the underlying cardiovascular or cardiorenal mischief. It is the latter which requires therapeutic attack. Another important consideration is that remedies acting upon the normal individual (or animal) may have an entirely different effect in hypertensive disease. This, I believe, is the main reason for disappointments in

the attempt to reduce blood pressure by vasodilators. They have been used innumerable times to decrease hypertension, in most cases with no or only slight temporary results. Exceptions will be noted later. Their inefficacy should be ascribed to the type of the pathological change underlying hypertension. For example, arterioles considerably thickened by disease, or in a state of hypertonus, can scarcely be influenced by drugs that exert their dilating power upon pliable arteries or under normal conditions of nerve tone.

In some hypertensive cases, there occasionally occurs a temporary sharp rise of blood pressure beyond the usual level for these individuals. This rise is sometimes marked by dyspnoea, precordial pain, headache, and vomiting. It is in these that relief from abnormal hypertension is especially desirable.

The **drugs** usually employed in the reduction of blood pressure are nitrite of soda, amyl nitrite, nitroglycerin, spiritus ætheris nitrosi, erythrol tetranitrate, and mannitol. In urgent cases, amyl nitrite in 5 minim pearls may be administered. Regarding nitroglycerin, the doses usually prescribed, from $\frac{1}{200}$ to $\frac{1}{100}$ of a grain three times daily, rarely results even in temporary blood pressure reductions. If its administration is not followed by dizziness, I am in the habit of prescribing much larger doses, as much as $\frac{1}{25}$ of a grain three times a day. In an experimental series of cases, for example, I have given as much as $\frac{1}{10}$ of a grain hypodermically. In those with uremia, the effects upon the blood pressure were disappointing. Even these large hypodermatic doses only had an occasional or temporary effect upon the blood pressure or symptoms. In others of this series, no constant result following nitroglycerin could be determined. Sodium nitrite, when efficacious, seems to have a more lasting effect. Erythrol tetranitrate has also been used with indifferent results. I have observed the best results when the vasodilators were employed in those individuals whom I have grouped as "hypertension and myocardial insufficiency with labile vasomotor mechanism" (*q.v.*). These patients are rarely uremic; the hypertension is relieved not only temporarily but sometimes for a prolonged period.

The action of **iodide of potash**, a drug frequently prescribed for the relief of hypertension, is still in dispute. In view of our knowledge that syphilis is a frequent cause of cardiovascular disease, its occasional good effects are to be ascribed, I believe, to its action upon the underlying luetic disease.

Venesection in all cases of hypertension with urgent symptoms is an excellent temporary therapeutic measure, especially in plethoric and cyanotic patients. From 400 to 800 c.c. of blood should be withdrawn.

Hydrotherapy — hot packs, oxygen-, carbon dioxide-, or hot baths, or the electric light bath — has been advocated to reduce hypertension. In so far as they promote diuresis and perspiration, and thus rid the

body of toxic material, they may have some direct therapeutic value. Oxygen and carbon dioxide baths are followed by varying and inconstant effects upon the blood pressure (Chapter XVI). The indications for these gas-impregnated baths in hypertension depend upon the degree of decompensation, and, in the main, follow those indications already discussed for that condition. If decompensation is extreme, baths are contraindicated; if mild, and observation shows good effects upon the blood pressure or symptoms, they may be continued. Even in those in whom there has been a reduction of blood pressure in or immediately following the baths, the reduction is slight and transient, so that the beneficial effect is probably to be ascribed to an action upon the general circulation rather than upon the blood pressure.

The effect of electric light baths, when efficacious in the reduction of hypertension, may be ascribed to the heat, incidental rest, and enforced quiet which form part of the treatment.

Diathermy — a special application of the D'Arsonval current to the precordium — is another method employed as a therapeutic agent in hypertension. Some of the reports of its effects are exceedingly enthusiastic; claims are made, for example, that hypertension is permanently relieved. In some cases which I have observed, in whom diathermy was used by other practitioners, the blood pressure remained at its usual level; there was no change whatever in the symptoms.

Percussion over the seventh cervical vertebra has been used, the object being to exert a reflex influence upon the vasodilators and thus upon the blood pressure. The effect of this procedure is, to say the least, exceedingly problematical and hypothetical.

Absence from business and treatments at Spas have undoubted marked therapeutic value in many cases of hypertension. The reasons are obvious. Patients are away from their ordinary environment, their routine of life approaches a more normal and physiological standard, excitement and nervous tension become almost negligible factors.

The amount of exercise that should be allowed depends chiefly upon the accompanying cardiovascular disease and upon the symptoms, rather than upon the degree of hypertension. At no time, however, should violent or fatiguing exercise be permitted. The amount of rest is also based upon the same criteria. For example, an otherwise active business man still capable of some physical exertion, who frets and becomes irritable when absolute rest is enjoined, can wisely be permitted to attend business for a short period each day. The week's work should be broken by one or two days of rest; or if the patient's condition allows it, by mild exercise out of doors, especially walking. Exercises of any type should be controlled by the state and degree of cardiac compensation, and by careful observation of the effect of exercise upon the individual.

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CHAPTER XX

"WEAK" HEART

Clinical Symptoms. — "Weak," "asthenic," "neurotic," "neurasthenic" hearts are some of the terms more or less loosely applied to various ill-defined conditions in which the salient feature is an unstable state of the vasomotor mechanism. I shall not here include a discussion of the arrhythmias which in themselves are sometimes regarded as evidence of a "weak" heart. Stress is often laid upon the presence of a soft, faint, systolic murmur over the apex, only slightly or not at all transmitted. Moderate hypotension (Chapter XIX) is also common in these individuals. Unless one chooses to interpret such findings as organic there is no evidence of organic cardiovascular disease. Subjectively, the patients complain of tiring very easily; if the occasion demands it, however, they can undergo long-continued physical exertion with no sign of strain upon the circulatory system. They also often complain of feeling faint or dizzy; sometimes they actually lose consciousness. Their faces and hands readily redden or blanch, with a corresponding feeling of warmth or cold in these parts. These changes are due to **vasomotor instability** and not to "weak" circulation. This is evidenced by the fact that these patients never suffer from edema or visceral congestion, or from any of the signs found in decompensation.

Aside from symptoms and physical signs referable to the circulation, these individuals sometimes suffer from ill-defined gastric complaints of non-organic nature often associated with referred intercostal and precordial pains. It is because of these that one's attention is drawn to the heart as the presumed offending organ. Nervous strain, joy, worry, excitement, and physical fatigue quickly elicit many of these symptoms I have referred to. The same factors seem occasionally to exercise an influence upon the apical murmur, which, at such times, becomes somewhat louder. The cause of this increased intensity is not clear; it may consist in some disturbance of the muscular ring at the mitral opening, allowing regurgitation.

These patients are as a class usually regarded as neurasthenic, because, though frequently of robust appearance, little or no physical basis for their symptoms can be found. Many have been fluoroscoped

in a search for some abnormality in the size and shape of the heart in order to account for the instability of the circulation. "Drop" hearts (Chapter IX) and abnormally small hearts (so-called microcardia) have been found in some of these individuals. I have studied a number of cases of "weak" hearts fluoroscopically. In some I have found the abnormal orthodiascopic types above referred to; in others, I have found the heart of normal size and contour, or with the left ventricle lying quite broad and flat upon the diaphragm. In other words, there was no orthodiascopic picture that I observed to be definitely correlated with "weak" heart. In addition, I have made the fluoroscopic observation that some of these "weak-hearted" individuals show particularly vigorous and strong ventricular contractions. It is therefore clearly evident that there is no constant parallelism between the vasomotor symptoms and the muscular power of the heart.

A few brief illustrative clinical histories and findings will serve to typify some of these patients.

A buxom woman of 45 had been told for years that her heart was 'weak.' There was no history of any previous serious illness. She had had two children who died. Very soon after the death of the last child, there began a series of symptoms consisting of giddiness, nausea, and flushing or pallor of the face. These symptoms have been intensified since her menopause two years ago, so that, in addition, she often feels faint and, in fact, actually fainted several times. There is a very soft systolic murmur at the apex, the orthodiascopic tracing shows an outline slightly broader than the normal, the blood pressure and all the other physical findings are normal. This patient has been under my observation for several years. She has undergone a severe operation for appendicitis with no effect upon her circulation.

A vigorous woman of 40, married and the mother of two children, while abroad was suddenly called home by illness in her family. She became worried, and soon complained of feeling fatigued; her hands and face readily became cold; she had pains across the chest upon exertion. There was a faint systolic murmur at the apex; orthodiascopic examination revealed a somewhat broad left ventricle; otherwise the cardiovascular and general examination revealed nothing abnormal. Upon being assured that her heart and other organs were normal, she soon recovered her mental poise. She began taking active exercise and now walks several miles daily without cardiac or other complaints.

A tall, narrow-chested, and somewhat anemic youth of 20 complained of frequent flushes and a feeling of "heat" in the face. The lungs and cardiovascular system were normal. The only abnormal finding was a narrow and pendulous heart. Upon being told after the examination that there was no lesion of any kind in the heart and lungs, he began leading a more normal and athletic life with rapid disappearance of the vasomotor symptoms.

I believe it is worth while emphasizing that the fundamental abnormality of this entire group of patients lies in an instability of the vasomotor mechanism, the cause of the irregular flushes, pallor, dizziness, and faintness. Nerve shock of any kind is often the culminating factor initiating the more acute symptoms.

To a great extent, **therapy** lies in firmly assuring the patient that there is no organic disease, and that the symptoms can be cured or at least greatly alleviated. It is a diagnostic and therapeutic error to dismiss these individuals by telling them that they are "nervous," for the symptoms are real and usually beyond their control. The treatment sometimes requires patience and always careful individualization. The patient should never follow any form of exercise, no matter how slight, to the point of fatigue. If no improvement follows, a rest cure for a longer or shorter time may be required. Patients too intent upon business must decrease the number of hours and the intensity of their work. At no time should these individuals feel hurried at their work or even at their pleasures. Rest in the reclining position, getting up late, having breakfast in bed, long rest at night, mild balneotherapy; later, graded calisthenics, or exercise in the open (golfing, tennis, swimming), are measures which, appropriately applied and carefully selected, are of great aid to the patient in helping him regain his vasomotor equilibrium and in finally enabling him gradually to return to his accustomed duties.

Among **drugs**, I have found a combination of atropine sulphate (grains $\frac{1}{100}$ to $\frac{1}{50}$) with nitroglycerin (grain $\frac{1}{150}$ to $\frac{1}{60}$), three times a day before meals, of most value. Where hypotension is present, suprarenal extract is sometimes of value. In addition, strychnine may be helpful. If symptoms are intensified at menopause, ovarian extract (corpus luteum) may be tried. It must be stated, however, that because of the type of the fundamental disorder — instability of the vasomotor mechanism — the effect of treatment is sometimes disappointing to the physician and discouraging to the patient.

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CHAPTER XXI

PRECORDIAL PAINS OF CARDIOVASCULAR AND OF EXTRACARDIAC ORIGIN — ANGINA PECTORIS

Historical. — As early as 1772, Heberden described angina pectoris. Nothnagel ascribed it to spasm of the coronaries, with consequent local ischemic and nutritive changes in the musculature, a theory which has profoundly affected the literature of the subject. Huchard divided angina pectoris into true and false: that produced by effort he called true, that without effort, false angina. Mackenzie ascribed the fundamental cause of cardiac pains, a term which he prefers to angina, to exhaustion of the cardiac musculature. Osler described four types of angina pectoris: the reflex, neurasthenic, hysterical, and the Nothnagel.

Confusion of Terms. — The frequent indiscriminate use of such terms as "false angina pectoris," "angina sine dolore," "angina vasomotoria," "angina vera," etc., with no clear sense of their etiology or pathology, has added to the difficulty of comprehending the fundamental causes of precordial pains. This confusion in symptomatology and pathology, and the fact that precordial pains of similar distribution and character may be caused both by intra- and extracardiac disease, have led me to make a careful study of those patients in whom these pains, from whatever cause, were the only or the most prominent symptoms. It appears to me that the term "precordial pain" obviates much of the confusion of the nomenclature, and, when etiologically defined, gives rise to a fairly clear clinical conception of the underlying cardiovascular or other disease. In every case that I studied, a very careful history (the importance of which is often overlooked) was obtained. Besides the routine, the clinical examination often included phenolsulphonephthalein and sodium chloride excretion tests, chemical examination of the blood, and electrocardiographic and orthodiascopic tracings. The type, character, intensity, and distribution of the pain, the time of its appearance, its correlation with exercise, with digestive or other disturbances, and the presence or absence of Head's zones, were noted. After study of all data, an attempt was made to discover the probable fundamental etiologic lesion in the cardiovascular system (myocardium,

endocardium, kidneys, or arteries), or in a distant organ (usually the gastro-intestinal canal), and to gauge the extent and severity of the pathologic damage to be therapeutically attacked. In this manner, besides obtaining a rational view of the entire subject, one could fairly definitely pick out and group patients in whom therapy would be of no avail, as well as those in whom it perhaps offered a good outlook for relief or even symptomatic cure.

Studies of the **cardiovascular nerve supply** have shown that there are rich ganglionic and nerve plexuses which surround the heart and the root of the aorta (Chapter I) and which are formed by branches of the vagus and sympathetic. There are also ganglionic cells and nerve fibrils in the sino-auricular node, the auriculo-ventricular bundle, and throughout the cardiac musculature (Chapter II). So far as known, nerves of sensation are absent. Through the fundamental work of Sherrington, Head, and, later, of Mackenzie, it is known that a viscus, though not possessed of nerves of sensibility, may, when irritated, excite the corresponding visceral segment of the spinal cord; the latter then sends abnormal centrifugal impulses to the muscles, glands, etc., which in the skin give rise to abnormal sensations usually felt and denoted as pain. In cardiac disease the area ordinarily affected is the precordium. Depending upon the nerves involved, the intensity of the irritation, or possibly upon irradiation of centripetal impulses to other spinal segments, pain may spread to the entire chest, to both arms (especially the left), the fingers, neck, head, the interscapular region, the epigastrium, the abdomen, and even the thighs. Besides the precordium and left shoulder, the epigastrium is the favorite site for referred pains, a fact which often causes erroneous diagnoses, and mistakenly directs the therapy to the stomach. In severe cases, pain is usually sharp, lancinating or agonizing in character, and combined with the oft-described feeling of impending death. In milder cases, it is dull or aching, or there may be merely a feeling of oppression on the chest. Head's zones are sometimes present, usually over the precordium, more rarely in the epigastrium. Occasionally the pressure even of wearing apparel produces severe pain. The sensitiveness may be confined to the underlying intercostal muscles; deep pressure alone then elicits pain. It is sometimes possible in a general way to judge of the effect of therapy and of the progress of the disease by the amount of pain elicited upon superficial or deep pressure; when the progress is favorable, sensitiveness to pressure diminishes.

In this connection it is important to discuss the frequency of **epigastric pains** and of occasional epigastric Head's zones found in heart disease. The former are usually ascribed to an enlarged liver or to congested gastric mucous membrane. However, the pains are present when the liver is not enlarged, and necropsy reports show that such patients often had no congestion or disease of the gastric mucosa. Be-

sides, we possess no data which definitely correlate such presumed congestion with pain. From clinical manifestations, from disappearance of epigastric tenderness *pari passu* with decompensation, and from the intimate correlation of the nerve supply of stomach and heart, it seems probable that this type of pain is chiefly, if not entirely, the **result of referred nerve excitation from cardiac disease.**

Clinically and etiologically, precordial pain is divided into that due to **intracardiac** and to **extracardiac** disease. This distinction is at times difficult or even impossible, but the attempt at differentiation is of fundamental importance. For the purpose of clinical classification, the term "myocardial insufficiency" is here used as equivalent to cardiac failure or incompetency.

Commoner Causes of Precordial Pain. — The subject will be considered according to this outline¹:

- A. Organic cardiovascular disease:
 1. (a) Hypertensive cardiovascular disease with myocardial insufficiency. (b) Hypertension and myocardial insufficiency with labile vasomotor mechanism. (c) Uremic group.
 2. Myocardial insufficiency without hypertension.
 3. Acute rheumatic endocarditis and rheumatic endocarditic exacerbations.
 4. Endo-myocardial disease with general circulatory failure.
 5. Embolic infarcts in the main coronaries and their branches.
 6. Cardiac syphilis.
 7. Premature arteriosclerosis and cardiosclerosis.
 8. Senile arteriosclerosis and cardiosclerosis.
 9. Sacculated aneurism.
 10. Tabagism (?).
- B. Extracardiac disease:
 1. Gastro-intestinal disease (organic and functional).
 2. Esophageal disease (organic and functional).
 3. Crises of catarrhal pulmonary affections.
 4. Vasomotor disturbances at the menopause.
 5. Neuralgias (?) of unknown origin.
 6. New growths and disease of the mediastinum, spinal cord and bony structures of the chest; intercostal myalgia and neuralgia; pericarditis.

A. PRECORDIAL PAINS DUE TO ORGANIC CARDIOVASCULAR DISEASE

1. (a) **Hypertensive Cardiovascular Disease with Myocardial Insufficiency.** — The patients of this group probably represent the most frequent sufferers from precordial pains. The cause of hypertension has been ascribed by some to an increase of epinephrin in the blood, though careful experiments by Janeway and Park have not justified this assumption. Recent studies of blood metabolism have shown that chronic nephritis with hypertension is often accompanied by an increased amount of non-protein nitrogen in the blood, and by a diminution of blood

¹Some of these groups have already been discussed in the chapter on Blood Pressure (Chapter XXI) from the hypertensive standpoint.

alkalinity, — facts which may in the future offer promising fields for therapy. The chief cardiac changes are ventricular hypertrophy, usually left but sometimes also right; patchy, fibrous myocarditis; thickened aortal and mitral cusps; lime deposits on the first portion of the aorta; and atheroma and thickening of both coronaries. It must be remembered, however, that patients with similarly diseased hearts may have little or no precordial distress.

A brief case description will serve to fix the type: a systolic blood pressure of 190 mm.; a rough first and a sharply accentuated and bell-like second sound at the right base; evidence of marked left ventricular hypertrophy with a heaving apical impulse; urine with or without albumin or casts; very slight pretibial edema; dyspnoea on exertion or appearing suddenly at night; nycturia. The pains are usually dull, most marked in the precordium, and radiate to the neck and arms.

Almost frantic therapeutic efforts are made, as a rule, to reduce the high blood pressure, while the fact that it is often a conservative, compensatory process seems to be entirely ignored. A mere enumeration of the long list of remedies is sufficient proof of their inadequacy. Vasodilators (nitroglycerin, amyl nitrite, erythrol tetranitrate), hot, Nauheim, and oxygen baths, violet ray, diathermy, and electric light baths are the most popular. There are conflicting and contradictory reports regarding all. In some cases, as already remarked (Chapter XIX), I studied the effects of hypodermic injections of 1 per cent solutions of nitroglycerin in doses of one tenth grain, three times a day, with no effect on the symptoms and only occasional temporary reduction of the blood pressure. The physical changes in the coronaries probably account for the futility of vasodilators to regulate the impoverished cardiac circulation and to relieve the resultant precordial symptoms. While theories such as cardiac spasm and anemia have had vogue as the causes of the pain, it seems more probable that the underlying factor is nutritional cardiac disturbance either from inadequate coronary circulation or, possibly, from toxic products flowing in the general circulation. Acute violent pains call for morphine. Pearls of nitrite of amyl may also be of aid. With milder symptoms, nitroglycerin occasionally gives some relief, especially if combined with atropine sulphate. Aside from temporary therapeutic measures, I have placed main reliance on digitalis, either digipuratum, or the tincture in 15-drop doses, three times a day. The drug should be given whether the auricles fibrillate or the pulse is rhythmic, and should not be stopped when pains and other symptoms improve, but continued in smaller doses for a long period. A curb should be put on the patient's physical activity. Mental excitement and stress should be avoided. The theoretical objection that digitalis may perhaps cause or increase coronary spasm has not been verified by clinical evidence in cases in which the drug had been given for months. In only one of my cases were

precordial pains increased by digitalis, although even here there was improvement for several months.

The importance of treatment of edema and other manifestations of cardiovascular disease lies in their fundamental association with precordial pains. This is illustrated in the following case:

A vigorous woman of 43, never pregnant, complained for three years of dyspnoea on walking, and stabbing precordial pains radiating to the left shoulder. The Wassermann blood reaction was negative. The urine contained a trace of albumin and, occasionally, hyaline casts. There was slight pretibial edema. Physical examination revealed typical signs of aortitis. The orthodiascopic tracing showed an enlarged left ventricle and a dilated aortal arch. The precordial area was tender to deep pressure. The lowest systolic blood pressure was 175 mm., the highest, 220 mm. There has never been any correlation between the symptoms and blood pressure. For the first few months she was put on digitalis, theobromin sodium salicylate, and occasional Karrell days. Within two months the cardiac pains and pretibial edema disappeared. Later, when remiss in taking medication and diet, dyspnoea and pain recurred. Under stricter surveillance, and the same medication combined with absolute rest at home on the Karrell days, twice weekly, she again slowly improved. This plan of treatment has been followed for three years. The blood pressure is still high; data revealed by physical and roentgen examination are the same, but there is very marked clinical improvement; precordial pains and edema have entirely, and the dyspnoea almost entirely, disappeared.

(b) **Hypertension and Myocardial Insufficiency with Labile Vasomotor Mechanism.** — In a smaller group of cases, in which the highest systolic blood pressure was around 180 mm., with marked diurnal variations of as much as 30 mm., precordial pains following exercise were the main symptoms. Nephritis was apparently not the main or the only cause of hypertension; emphysema and myocarditis were the chief pathologic conditions. Experimental subcutaneous injections of nitroglycerin in doses of one fiftieth grain three times a day had a marked temporary effect on the blood pressure, and, usually, on the symptoms; in one instance, the injections were regularly accompanied by sudden relief of precordial pains to be followed by giddiness. Such cases apparently represent examples of disturbed labile vasomotor mechanism rather than hypertension due to marked vascular disease. Nitroglycerin or other vasodilators given at the onset of pain are apt to be followed by great relief. Digitalis, though useful, is not as beneficial as in Group 1.

(c) **Uremic Group.** — Headache, nausea, vomiting, varying grades of anemia, attacks of paroxysmal dyspnoea and of precordial distress, high systolic and especially diastolic pressure, nocturnal polyuria, and changes in the retina, are the cardinal symptoms. Kidney test meals, as advocated by Schlayer and others, usually show decreased salt, nitro-

gen, and water elimination; and the blood, retained non-protein nitrogen. The facial appearance in typical instances is characteristically pallid or ashy gray.

The precordial pains are not relieved by nitroglycerin, digitalis, or theobromin sodium salicylate. They are apparently caused by toxic products flowing in the general circulation and affecting cardiac nutrition. Dietetic measures, especially a diet low in protein and rich in carbohydrates, are of most value. When renal tests show no water retention, ingestion of large quantities of fluids is sometimes beneficial. For the relief of pain, occasional doses of morphine are necessary. On the theory of tissue acidosis, I have tried the intravenous injection of 500 c.c. of a 5 per cent bicarbonate solution in two cases. In one, there was marked relief of the precordial pains and other symptoms; in the other, the pain was relieved only for some hours. Glucose solutions given by the Murphy drip or even intravenously may be of value.

2. Myocardial Insufficiency without Hypertension.—The main complaint of these patients, usually men of sedentary habits between the ages of 50 and 60, is slight precordial pain following exertion. The patients look very well preserved. The urine is normal or may contain a slight trace of albumin with a few casts. Physical examination reveals a slight impurity of the first sound at the apex, and a soft systolic murmur at the base; there is no evidence of severe cardiovascular disease. The systolic blood pressure is around 160 mm., rarely much higher; there is no edema. The orthodiascopic tracing usually shows a slightly dilated aortal arch with the left ventricle lying broad and flat on the diaphragm; it is impossible to state if this ventricular contour is due to flabbiness or to hypertrophy. It is interesting to note that these patients commonly give a history of having had painful gastric attacks in previous years, with symptoms pointing to gastric or duodenal ulcer. Excellent results have followed the use of digitalis, given at first in large, and then in moderate doses, and continued intermittently for weeks or months. Small doses of atropine were sometimes added. At the beginning, the patient's activity was somewhat restricted; later, moderate exercise—golf or walking—was advised. Acidulous food and drink were interdicted, and some light form of nourishment between meals was prescribed. The following is an illustrative case:

Male, age 60, has for years been careful about his diet, particularly in the avoidance of acids, for indiscretions were followed by epigastric pains. For several months he has complained of precordial, knife-like, paroxysmal pains on walking. Examination shows a well-nourished man; there is no edema; the urine contains no albumin or casts, the average systolic blood pressure is 160 mm. The orthodiascopic examination reveals a somewhat enlarged left ventricle, the aortal outline somewhat broader than normal. There is an impure first sound at the apex and at the base. There is no dyspnoea or de-

compensation. One digipuratum tablet was given, at first three times daily, and then continued in smaller doses for several months. Atropine sulphate, $\frac{1}{160}$ grain, three times a day, was also prescribed. The patient is now doing his regular work, has taken ocean baths during the summer, and has had no recurrence of precordial pains.

3. Acute Rheumatic Endocarditis and Rheumatic Endocarditic Exacerbations.—These cases usually occur in young persons with definite rheumatic histories, with mild tachycardia, with no dyspnoea, and with marked auscultatory evidence of valvular disease (usually mitral stenosis). Slight and irregular rise of temperature is the rule. The patients complain not only of the subjective feeling of palpitation, but also of "sticking" pains localized in the region of the heart. There are usually no Head's zones. The cause of the rapid heart action and of the precordial pains, even in the absence of abnormal temperature and other rheumatic manifestations, appears to lie in the irritative effect of fresh exacerbations of endocarditis. The best medication is sodium salicylate given in full physiologic doses. My routine has been 15 grains hourly until six doses have been given or tinnitus occurs; the dose is then decreased. Bromides in moderate amounts, and ice bags to the precordium, are also helpful. Comparative or absolute rest in bed may be necessary for some time.

4. Endo-myocardial Disease with General Circulatory Failure.—The fundamental cause of the pains is apparently nutritional disturbance from local circulatory failure in the heart itself. Precordial Head's zones or muscle tenderness are often present. The therapy for the relief of pain is the same as for the decompensation. This ordinarily means vigorous and long-continued digitalis medication, and, for the relief of edema, theobromin and the dietetic regimen already outlined. If compensation is restored, cardiac pains and Head's zones disappear.

5. Embolic Infarcts of the Main Coronaries and their Branches.—It is known that patients who have recovered from cardiac pains have occasionally shown myocardial scars and other changes resulting from old infarcts. Huchard, in a summary of 145 necropsies of coronary disease with cardiac pains, found five due to embolism of the artery. Recently several cases with necropsy reports of embolic infarcts of the main coronaries have been described; the patients died within a few days or hours with symptoms of intense precordial distress. I have observed several cases with intermittent pains lasting days or weeks, in which the symptoms were possibly caused by infarcts or emboli of the smaller coronary branches. Curschmann in 1891 first described this condition; he reported three cases with necropsies. Two patients died some years after the onset of symptoms. Both showed localized myocarditis. In one, there was aneurismal dilatation confined to one sclerosed arteriole; in the other there was an obliterated coronary branch of the third order. The third patient, in whom the condition

was correctly diagnosed, died suddenly; an embolus was found in a coronary branch of the second order.

The cases I observed are:

Female, unmarried, aged 18, under observation three years, gave a typical rheumatic history. There is a marked double aortic lesion and a tremendously hypertrophied heart. The Wassermann reaction and frequent blood cultures were negative. The systolic blood pressure was 180 mm., the diastolic, 20 mm. For months she has had many attacks of moderate irregular fever. On several occasions they were initiated by sharp precordial pains followed by tachycardia or auricular fibrillation, convulsive twitchings and tremors, and by loss of consciousness lasting several days. Latterly these attacks have become more severe and were followed by well-localized Head's zones.

Female, unmarried, aged 20, gave a history of continued attacks of rheumatism and "heart trouble." During the last few months she had two attacks similar to but not quite so severe as that with which she entered the hospital. There were continued agonizing pains and exquisite tenderness, even to the slightest touch, over the precordium, and radiating pains to the left shoulder and forearm. The temperature was 104°. A mitral regurgitant lesion was present. There was no edema. Breathing was frequent and shallow, apparently owing to the attempt to keep the chest at rest. After one week, high temperatures, sharp pain, and rapid breathing disappeared; the patient felt comfortable, but precordial pain on firm pressure was still present when she left the hospital.

Male, aged 40, with a double mitral lesion, gave a history of having felt well until three months prior to hospital admission. He then had an attack of mania (?) lasting three days. Subsequently pneumonia and pleurisy developed, accompanied by chills and high fever, and by attacks of paroxysmal auricular fibrillation. After several cultures a non-hemolytic streptococcus was isolated from the blood. During several months of hospital observation there was a constant sensitive area near the cardiac apex.

The characteristic of these three cases was not only the rise of temperature and evidences of active endocarditis, but also the progressive tendency of the disease and the presence of local, tender precordial areas. Though these manifestations may have been due to the endocarditis alone, the distinct localized sensitive areas, and the clinical course, make it probable that, in addition, acute focal myocarditis was present. From clinical and physical signs, and from the progressive and probably bacterial nature of the endocarditis, it seems fair to assume that such focal myocardial changes originated in embolic infarcts of the smaller coronary branches, accidents not necessarily incompatible with life.

Necropsy findings will of course be necessary to establish the diagnosis in such cases.

6. **Cardiac syphilis** is an extremely frequent cause of precordial pain. It is usually substernal, dull, boring, and aching; but may, however, have the distribution and characteristics of the types already described. One can probably ascribe the frequency of these substernal pains to the almost invariable presence of syphilitic aortitis and peri-aortitis with consequent dilatation, to the fact that the root of the aorta is surrounded by rich ganglionic and nerve plexuses, and to the varying degrees of aortic dilatability and pressure. Head's zones are comparatively rare. **Therapy** consists in the treatment of the underlying disease. Although salvarsan was originally considered contraindicated in cardiac syphilis, abundant experience has since shown that, given first in smaller, later in larger doses, salvarsan combined with the usual mixed treatment is of great and definite value. The best routine method of its administration is 0.2 gm. injected intravenously every week until 0.6 gm. is given; later, full doses may be given, the frequency depending on the cardiac condition. Salvarsan sometimes benefits and controls the pains immediately, apparently because of reduction in the syphilitic inflammatory exacerbations. Where the pathologic process in the aorta, the coronaries, or myocardium has reached an extreme degree, salvarsan or any other treatment can be of little or no avail. However, since our present methods of examination are not sufficiently exact to diagnose such conditions, I believe the treatment outlined is indicated in every case of cardiac syphilis except when the patient is moribund. Cardiac failure, if present, should receive its appropriate treatment, that is, digitalis; and if necessary, theobromin sodium salicylate.

7. **Premature Arteriosclerosis.** — This comprises a rare group found in young adults. Persistent precordial distress is often present for months. Gastric symptoms similar to those of hyperacidity occasionally dominate the clinical picture. Physical examination may give no hint of the severity of the pathologic process attacking the entire cardiovascular system, and finally resulting in extreme changes throughout the aorta, coronaries, arterioles, and endocardium. Indeed, such hearts may be pathologically identical with those of persons dying of senility. For months there may be no decompensation in the ordinary sense. The urine may be normal, the blood pressure not high, the heart sounds somewhat distant, and the only indication of severe cardiac disease, besides the precordial pains, may be the presence of an arrhythmia, usually marked sinus arrhythmia, or extrasystoles. Other occasional symptoms are dyspnoea and nocturnal attacks of precordial distress.

An illustrative case with necropsy report follows:

Male, aged 42, of athletic build, had been a heavy smoker. For four years he had suffered from gastric symptoms resembling hyperacidity; belching was particularly prominent. The gastric contents showed high values for free hydrochloric acid. He had frequent attacks of vasomotor disturbances: numbness, coldness, and pallor of the hands. It

was only late in the disease, when an attack of hemiplegia occurred, that attention was focused on the possibility of a generalized arteriosclerosis as the underlying disease. Although several Wassermann tests were negative, salvarsan and mixed treatment were given; these were without effect. At necropsy, marked thickening and calcareous deposits were found on all the cardiac valves; the coronaries and their branches showed extreme thickening; the myocardium presented many fibrous patches; the entire heart was somewhat enlarged. Spirochetes were not found in the aortal tissue. The heart accurately resembled that of a very old person suffering for many years from severe generalized arteriosclerosis and cardiosclerosis.

The **cause** for such presenile sclerosis is still undetermined. It is possible that unknown infections and toxemias are etiologic factors. In one of my patients the disease originated within two years of an obscure pulmonary infection of several months' duration. For the present, one can only state that, from some unknown cause, the elastic tubing comprising the vascular system becomes prematurely defective and diseased. Except digitalis for temporary relief of dyspnoea, treatment is of no avail.

8. Senile Arterio- and Cardiosclerosis.—The clinical picture is usually clear. All the palpable arteries are tortuous and thickened; the blood pressure is normal or not very high, the heart somewhat enlarged; the systolic sounds at the apex and right base are impure; the second sound at the right base is sometimes accentuated. The precordial pains usually occur with exertion; although sometimes intense, they are more often of mild character. Apparently they basically depend on coronary sclerosis. Because of the advanced pathologic changes the patients can rarely be relieved by medication. Digitalis and theobromin sodium salicylate sometimes help, the amount of relief apparently depending upon the degree of healthy tissue still remaining to react to medication. The patients usually require prolonged periods of comparative rest.

9. Sacculated Aneurisms.—In a general way, the precordial pains depend on the size and position of the aneurism. If large, the pains are those due to pressure on the surrounding structures (intercostal nerves, ribs, etc.). If small, and involving the first portion or arch of the aorta, the symptoms are similar in character and etiology to those described under cardiac syphilis (Group 6). The therapy is also the same as that there indicated. My experience with wiring and the electric current treatment according to the method of Lusk is limited to two cases. In one, after some weeks, small, frequent external hemorrhages occurred, due to the perforation of the sac by the end of the wire; the patient finally died of anemia. In the other, the patient did not improve during his stay in the hospital; he died suddenly some weeks later; a necropsy was not permitted.

10. **Precordial Pains Due to Tabagism.** — It is questionable whether the "*tobacco heart*" should be classified under organic cardiovascular disease. Since it has been shown experimentally that intravenous injections of nicotine into rabbits are occasionally followed by aortal atheroma, it has by some been assumed that smoking in man may produce similar changes in the vascular system, and possibly in the myocardium. Direct proof, however, of such damage is still lacking. Besides, it has been shown that atheroma is sometimes found in the normal rabbit aorta. In a number of experiments by Gy. in which rabbits were made to inhale smoke for a definite period of time, only a very few were found to have aortal changes; indeed, the proportion was not more than that usually in the normal animal. In a study of a large series of hearts of smokers who died from other causes, one observer found no changes except slight degeneration of the papillary muscle ascribable to the rapid heart action usual in smokers. Experiments have shown that nicotine, the main tobacco alkaloid, is a powerful neurotropic poison affecting particularly the sympathetic ganglia and the peripheral vasodilators. In view of these observations, and in the absence of proof of pathologic changes in the human heart due to tabagism, it is reasonable to assume that the frequent precordial pains and distress are caused by disturbance in the coronary circulation, and by referred pains from irritation of the sympathetic ganglia.

The pains may be dull and aching, or sharp and radiating; occasionally, the first premonition is very sharp lancinating precordial pain radiating to the left shoulder and forearm, and accompanied by unconsciousness. For example, a healthy man of 40, a heavy smoker, while running for a train, was suddenly attacked by terrific precordial pain followed by unconsciousness for one hour. The cardiovascular examination revealed nothing abnormal. For weeks after the attack, any slight movement — even turning in bed — brought on pains. The patient was finally able to be about and resume his usual occupation. At present, six years after the first fainting spell, he feels quite well. He again smokes, but moderately.

The most frequent of the **arrhythmias** in smokers are extrasystole, usually auricular. Other arrhythmias, however, are occasionally encountered. Thus I have seen two cases of sino-auricular block (Chapter VII), one of auricular flutter and one of auricular fibrillation.

Most cases of tobacco pains and arrhythmias cease when smoking is stopped. Occasionally both recur intermittently for years. In such instances nitroglycerin given regularly, or during pain, is of benefit. The bromides are of value in helping to control persistent arrhythmias.

B. PRECORDIAL PAIN OF EXTRACARDIAC ORIGIN

1. **Gastric Disturbances.** — Patients with gastric disturbances, particularly those in whom epigastric distress is marked, are apt to suffer

from referred precordial pains. The clinical picture is that of hyperacidity, or of gastric or duodenal ulcer. Belching, loud and explosive in character, is usually a prominent symptom. The precordial pains are commonly sharp, neuralgic, fleeting, and inconstant in character; their distribution is usually along the fourth and fifth left intercostal spaces. Pain referred to the left shoulder is rare. Corresponding to the anterior distribution, there may be a similar area posteriorly. A precordial Head's zone is unusual; its area of distribution inconstant. There may be a close association between gastric symptoms and precordial pains, particularly when "hunger pains" and belching are marked. There is no decompensation or dyspnoea. When gastric symptoms are stormy, extrasystoles may occur. Treatment is naturally directed to the underlying disease. I have found most benefit from atropine given in full physiologic doses three times a day before meals, and from an antacid powder containing equal parts of sodium bicarbonate, magnesium oxid, and oleosaccharated peppermint in half teaspoonful doses after meals. Appropriate diet given frequently and in small quantities is important. As already noted, gastric symptoms also accompany cardiac disease, so that the correlation and study of all the clinical data are necessary for a correct diagnosis. Unless extrasystoles are subjectively annoying, they require no medication; otherwise they may be partly controlled by bromides.

2. Precordial Pains from Esophageal Disease. — Carcinoma, syphilis, ulceration, and diverticula are the usual organic causes of referred intercostal and precordial pains of esophageal origin. In some cases, organic disease is absent; the symptoms may then be due to esophageal spasm, especially in the region of the cardia. The usual symptoms are an uncomfortable, conscious gulping effort on swallowing solids or fluid, accompanied by sharp pains referred to the lower sternum, and radiating to the precordium or even to the upper and lower extremities. Such symptoms are occasionally mistaken for those due to aortic aneurism; and the difficulty in swallowing, to aneurismal pressure on the esophagus.

An illustrative case with therapy follows:

Male, aged 48, stated that for seven years he had "choking sensations" in the larynx and severe substernal pains on attempting to swallow solid food. These pains occasionally radiated to the head, arms, and legs. The symptoms became progressively worse. The neurologic and physical examination revealed nothing abnormal; the fluoroscope showed a normal aorta. Wassermann reactions of the blood and spinal fluid were negative; the stools, urine, and gastric contents were normal. Roentgenograms showed a pouch-like dilatation of the middle part of the esophagus. An esophageal bougie was arrested opposite the middle of the sternum. Finally a small bougie was passed. For purposes of dilatation bougies of increasing caliber were employed. At first semi-solid, later solid food was given. Atropine sulphate, $\frac{1}{100}$ grain three

times a day, was administered. The pains gradually subsided and finally disappeared. The patient was taught to pass a stomach tube; this he did for some weeks after he left the hospital. An examination months later showed that esophageal symptoms and pains had disappeared, and there had been a considerable increase in weight.

3. Precordial Pains Accompanying Crises of Acute Pulmonary Affections.—Such attacks of precordial pain are fairly common with the crises of influenzal bronchitis, especially when sharp critical sweats are present. Head's zones in the left nipple region, and arrhythmias (especially sinus arrhythmia and extrasystoles), are frequent, a combination suggestive at first sight of toxic myocarditis. Symptoms, however, last only a few days; except for pain and arrhythmia, the heart is normal; there is no decompensation or edema. Examinations made months or years later have shown that the cardiovascular system had not been damaged.

The following is an example:

A physician, aged 43, never had any cardiac complaint. He contracted pharyngeal and bronchial grippe. There were critical defervescence, sharp sweats, an irregular pulse, and sharp stinging pains in the left breast. The patient feared that he had an infectious myocarditis, and carefully avoided every unnecessary exertion. Examination revealed a small Head's zone confined to the left nipple region, and an arrhythmia which polygraphic and electrocardiographic tracings showed to be due to auricular extrasystoles. Otherwise the cardiovascular system was normal. The patient was reassured, told to get out of bed, and given atropine sulphate, $\frac{1}{150}$ grain three times a day before meals, and bromides at night. He was sent to the country and advised to exercise as much as he chose. He was soon able to walk several miles daily. Within one week the extrasystoles and pain disappeared, and have not since returned.

4. Precordial Pains Accompanying Vasomotor Disturbances at the Menopause.—Women at the climacteric period, with marked vasomotor disturbances (flushes, heat flashes, cold extremities, etc.), frequently have persistent precordial pains without evidence of organic disease of the heart or neighboring organs. They are possibly evidence of vasomotor circulatory disturbances in the heart itself. The patients rarely react well to medication. Bromides and small doses of atropine and nitroglycerin are of most value. Ovarian (corpus luteum) tablets are occasionally helpful. Hurry and excitement should be avoided.

5. Precordial "Neuralgias" of Unknown Origin.—Under this heading is grouped a small number of individuals, usually young and apparently healthy and vigorous, without vicious habits, with no rheumatic manifestations, in whom continued and frequent examinations of the heart and other organs reveal nothing abnormal. Occasionally, sudden explosive belching or singultus occurs. In one of my cases, smoking some years previously might have been an etiologic factor.

The pains are apparently quite haphazard in their onset and duration, and in my experience defy medication and therapy.

In this group may also be placed females who complain of indefinite chest pains and distress, and who, from other manifestations, apparently suffer from a functional disorder of an internal secretory organ or organs. These patients are occasionally relieved by medication aimed at substituting the presumed secretory deficiency.

6. **Pott's Disease, Mediastinal and Spinal Tumors, Tabes, Inter-costal Neuralgia and Myalgia, Pleurisy, Pericarditis.** — These are additional extracardial conditions and diseases producing precordial pain.

The etiology and types of precordial pains here described are admittedly incomplete. Only those frequently encountered and clinically important have been discussed, and their correlation with intra- or extracardiac disease described. It is recognized, also, that the various groups sometimes overlap. In organic cardiovascular disease, I have endeavored to estimate roughly the extent and nature of the pathologic damage, and to gauge, if possible, the amount of remaining healthy tissue which may be favorably influenced by therapy. Thus studied, one obtains a basis for rational therapy, with its prospects and limitations. The postmortem specimens of advanced coronary, endocardial and myocardial disease, with the history of precordial distress during life, should not blind us to the fact that some of these patients have lived many years despite their diseased organs.

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CHAPTER XXII

THERAPY OF PNEUMONIA FROM THE CIRCULATORY STANDPOINT

BECAUSE of the frequency with which cardiac drugs are employed in pneumonia, I wish to review my impressions gained in the drug treatment of many cases of pneumonia. I shall not touch upon the bacteriological or chemotherapeutical aspects. Statistical study of various epidemics or methods of treatment have likewise not been dwelt upon. Indeed, it is because I feel that none of the methods of medicinal treatment has been followed by any demonstrable correlation of cause and effect, or of therapy and subsequent cure, that I have discarded statistics as bringing us no nearer to the therapeutic and circulatory problems of pneumonia. A study of these methods, some of them directly antagonistic in theory and practice, arouses doubt as to their possible efficacy. Mere mention of some of the types of treatment, many long since discarded, may therefore not be amiss. Creosote, alcohol, no treatment until the crisis, no treatment for hyperpyrexia, cold sponges or even tubs for the same symptom, no stimulation, stimulation at the crisis only, stimulation to prevent the dangers of the crisis, camphor in oil, caffein, digitalis, adrenalin, venesection, fresh air, cold air, are or have been popular.

"**Stimulation**" has been the usual therapeutic keynote, for the danger was always correctly conceived to be circulatory failure. I have used all of the recognized cardiotonics — digitalis, strophanthus, caffein, adrenalin, as well as those whose effect upon the circulation may well be questioned — alcohol, strychnine, camphor. Camphor I have used in smaller, repeated, as well as in several larger hypodermic injections, though experiments have shown that camphor in oil had no effect in delaying or preventing death in artificially induced pneumonia. I have employed the other drugs mentioned in all possible forms and combinations; in heroic, in small and moderate doses, subcutaneously, intravenously, and internally, before, during, and after the crisis. With possible isolated exceptions, I have yet to see any of the effects upon the circulation which follows the use of some of these drugs in the experimental animal, or in cardiac failure from cardiovascular disease.

The chief reasons for disappointment in pneumonia therapy are, I believe, that we are dealing with an infection of varying virulence and involving varying amounts of lung tissue. Therefore I have attempted, for clinical and therapeutic purposes, to roughly group and to treat pneumonia according to **degrees of toxicity** and the **extent of lung involvement**. I consider the **crisis** a separate problem for therapy.

1. **Toxic Cases.** — In the extremely toxic group, typically exemplified by sharp onset, early delirium, subsultus, dry tongue, no pain, rapid pulse and breathing, my experience has been that attempts at "stimulation," a term which we shall apply to drug therapy used to combat and treat circulatory failure, are almost always without efficacy. These patients apparently defy all drug therapy and sometimes die before the area of pneumonic involvement becomes clinically recognizable. Death ensues from acute toxemia. Stimulation, even if heroic, has not in my hands in any visible manner delayed or prevented the fatal outcome. The reason is apparently because the drugs do not combat the infective factor. Experiments throw an interesting sidelight upon this problem. It has been found that hearts perfused with pneumonic blood lose their efficiency, which is later restored by the use of normal blood. Whether in the human being the changes wrought in the cardiac musculature are, or later become, myocarditis are questions into which we shall not enter.

It is usually believed that in toxic pneumonia there is marked vasomotor failure, though others, as the result of animal experimentation, have held otherwise. I have only very rarely seen any therapeutic result from single or repeated injections of adrenalin given subcutaneously or intravenously. A like negative result has almost regularly followed the use of strophanthin and other digitalis bodies when the pulse was regular. That digitalis does localize itself and affect the heart muscle in pneumonia as in purely cardiac disease has recently been shown by A. E. Cohn, who found the usual electrocardiographic evidence of digitalization — a negative *T* wave (Chapter VI). Despite this change in the electrocardiogram, I have found, in rhythmic cases, no evidence of any help to the circulation from digitalis — cyanosis was not decreased, dangers from edema of the lungs not reduced.

As a favorable prognostic sign, Gibson has emphasized a certain parallelism between pulse rate and systolic blood pressure; but others as well as myself have found his so-called law of no significance. The removal of 500 to 700 c.c. of blood by venesection gives occasional temporary relief in toxic cases; the benefit seems attributable to ridding the body of toxic material rather than from any primary circulatory relief.

2. **Area of Pneumonic Involvement.** — With no evidence of severe toxemia, slowly progressing consolidation confined to one lung is usually not dangerous during the acute febrile period. Such are probably the cases in which no treatment or any treatment has its measure of success.

If, however, there is massive sudden involvement of one entire lobe and part or all of another, the inflamed area in itself offers a problem for therapeusis, aside from the question of toxicity and crisis; for the pulmonary circulation may be sufficiently interfered with to produce cyanosis and beginning circulatory failure. Here venesection is clearly indicated for the relief of pulmonary stasis; "stimulation" has a better chance for success, for the problem approaches more nearly a purely circulatory one. Cardiac tonics should be given in large doses from the very outset, to combat, if possible, ever impending circulatory failure.

3. The **crisis** apparently marks that point at which there is more or less complete sudden destruction of the pneumococci in the lung, while lysis marks their more gradual destruction. The main dangers during crisis are **general**, from sudden systemic toxic invasion; and **local**, from edema of the lungs. The local danger, from the circulatory standpoint, depends upon the amount and rapidity of resolution, and consequent pulmonary edema. An extremely important factor, scarcely mentioned in the literature, is the presence or absence of expectoration at the crisis. Pulmonary edema complicates and accompanies resolution more frequently if expectoration is absent; while if expectoration takes place, there is increasing opportunity for pulmonary aëration, and less for edema. I always insistently try to make the patients cough in the hope of loosening and expectorating the pneumonic products. When, despite this, edema of the lungs supervenes, as shown by the loud mucous bubbling râles over the pneumonic and other areas of the chest, the chances for success become less, but even then I have the attendant regularly rouse the patient to coughing efforts as long as consciousness is retained. As far as one may judge, stimulation in acute pulmonary edema occasionally keeps the patient alive for some hours or even a day, and may exceedingly rarely seem to pull a patient from death.

With **lytic** or **gradual resolution**, even with very sharp critical drops of temperature, the battle is usually won, for edema of the lungs rarely occurs and stimulation may be efficacious. However, even in cases of non-massive pneumonic involvement in young, vigorous individuals, with no evidence of toxicity during the attack, sudden, almost tragic intrusion of critical resolution, edema of the lungs, and death — all within a few hours — may supervene. One instance especially impressed me — that of a young, healthy individual to whom the pneumonia was literally a laughing matter. From the presence of perspiration and from the physical signs in the lungs, I was able to foretell the onset of critical resolution some hours before the typical drop in temperature. Heart and pulse were then perfectly satisfactory. Herculean efforts were made by stimulation and otherwise to ward off the dangers of edema of the lungs. Within six hours, however, the patient died from pulmonary edema; while in an adjoining ward, an old woman, severely poisoned by her pneumonia and very sick for over one week,

had reached the crisis with slow, gradual resolution of the pneumonia, and was slowly and surely convalescing.

Where digitalis has been given, its effect in causing arrhythmia must naturally be considered. Aside from this, a study of cardiac **irregularities** occurring during the course of, and at the crisis of, pneumonia offers interesting and important therapeutic problems. The usual types are moderate bradycardia, sinus arrhythmia, extrasystoles, auricular fibrillation, and heart block. It is sometimes assumed that the presence of the arrhythmias is indicative of some organic affection of the cardiac valves, or of the musculature. But, as in the non-pneumonic individual, irregular heart action does not in itself necessarily mean heart disease. Indeed, some of these arrhythmias — especially sinus arrhythmia, moderate bradycardia, and extrasystoles — when found with an **unembarrassed circulation**, at or immediately after, the crisis usually offer good clinical evidence that the disease has definitely run its course. The irregular cardiac action is apparently due to a neurotropic effect of pneumonic toxins. Whether the latter act upon the medullary center or upon the heart itself it is impossible to state. Because of their occurrence at the time of crisis, and because some drugs (for example, morphine) produce arrhythmias by affecting the cardio-inhibitory center, I am inclined to the former view. These arrhythmias usually last a few hours or days. I have followed some of these cases for years and have never observed any correlation between the arrhythmias and the possible later development of organic cardiovascular disease.

I have observed auricular fibrillation — complete irregularity of the pulse — several times at the crisis. In three such patients, each of whom had several crises from successive involvement and resolution of various pulmonary areas, there were attacks of fibrillation lasting several hours with each crisis. Two of the patients were elderly; the third, a vigorous adult. None showed circulatory embarrassment or heart failure during the course of the fibrillation; on the contrary, there was the usual picture seen in favorable crises with normal rhythm. On the other hand, I have observed other cases of auricular fibrillation occurring during the course of toxic pneumonia, all of whom died. In these it was impossible to estimate how much the arrhythmia itself contributed to the dangers of the pneumonia.

Complete heart block with slow ventricular rhythm is rarely found in pneumonia. That it may be functional in origin and probably due to abnormal action upon the cardio-inhibitory center seems indicated by two of my cases, one of which came to necropsy. The patient had been ill for some time, there was no history of digitalis medication. Microscopically and grossly, the cardiac musculature and the bundle of His were found normal.

The **therapeusis** of arrhythmias developing in pneumonia is similar to that in non-pneumonic individuals. If accompanied by heart failure, the arrhythmias are of serious, possibly ominous import. Except in

heart block with slow ventricular activity, when atropine should be tried, the treatment of these arrhythmias is no different from that of the other phases of pneumonia already discussed. If arrhythmias are unaccompanied by failing circulation, though they perhaps should not be entirely disregarded, their import is slight and they rarely require separate medication.

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